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AMERICAN JOURNAL OF SURGERY

A Magazine of Practical Surgery in all its branches

WALTER M. BRICKNER, M.D., F.A.C.S.
Editor
PUBLISHED MONTHLY BY SURGERY PUBLISHING CO.
ESTABLISHED 1905 BY J. MACDONALD, JR., M.D.
Publication Office: 425 Carroll St., Elmira, N. Y. Administrative and Editorial Office: 15 East 26th St., New York City, N. Y.
Copyright 1922, by Surgery Publishing Company.
Entered as second class matter, April 4, 1921, at the Post Office at Elmira, N. Y. under the Act of March 23, 1879.

VOL. XXXIX. FEBRUARY, 1925 No. 2

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AMERICAN
JOURNAL OF SURGERY

VOL. XXXIX. FEBRUARY, 1925 No. 2

LESIONS OF THE TIBIAL TUBERCLE (OSGOOD-SCHLATTER'S DISEASE)

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The purpose of this study is to report a series of seventeen cases demonstrating lesions of the tibial tubercle of the Osgood-Schlatter type.

The cases selected were seen during a period of three and one-half years in the roentgenological department of the Hospital for Joint Diseases. The majority were found in routine examinations of the knee in adolescents, reporting to the hospital for various injuries or ills, in and about that joint.

Reference in the literature to this type of affection dates from a contribution by Key in the *Lancet*, April 15, 1827, and one by DeMayer in the *Medical Times* in 1833. Following the publications of these papers, numerous articles appeared chiefly in the German and French publications. The writers in the main described this condition from a purely clinical and surgical aspect.

With the appearance of the roentgen ray as a diagnostic means, numerous investigators published their observations. The earliest reference in the American publications that discussed the subject from a roentgenological standpoint appears to be those of L. A. Weigel[1] and R. N. Lovett[2] in individual contributions in the *Philadelphia Medical Journal*, January 6, 1900. They published radiographic reproductions of knees showing the condition together with brief descriptive texts. No detailed roentgenological study, however, appeared until the work of Osgood[3] and Schlatter[4] in 1903. Working independently, they reported their observations at about the same time, so that today the disease is rightly named after them. Osgood's original description contained a report of 10 cases together with an anatomical dissection of two knees, with special reference to the mechanics of the production of the injury. Gaudier and Bouret[5] in 1905 brought the literature up to date.

Following these publications there appeared those of Kirschner[6], Bergmann[7], Kienbock[8], Dunlop[9], Graef[10], and Hodgson[11]. Bergmann and Kienbock were of the opinion that these lesions were due to some rachitic change, whereas Graef thought that many of these conditions were due to infection, probably by the staphylococcus. Dunlop's excellent contribution contains a splendid bibliography of the literature. He states—"The impression I have obtained from my studies and observations, leads me to believe that the borderline between the so-called inflammatory lesions of the pretibial tubercle and the actual separations of the tubercle, is one of convenience".

Saule [12] in 1916 described an operation for the fixation of the tubercle to the tibia. The following years were productive of many articles on the subject, among which were those of Stephens[13], Boder[14] and Gibson[15]. Stephens believed that the disease should be classified as an injury and not as a true disease and states that there is always an accompanying inflammatory reaction similiar to a periostitis.

The present conception of the disease is varied. As a result of the somewhat acrimonious debate, chiefly in the German literature, both infection and trauma have been postulated as causative factors. There are basic reasons for the support of both groups. To rigidly classify this condition as solely traumatic or solely infectious, is somewhat hazardous. We have cases that are classified in either group. We would carry Stephen's analogy somewhat further. As it is well known, a periostitis may be produced by local injury or local infection, or as a local manifestation of a general infection. We have then three causes, all different, each of which may be productive of a similar roentgenographic appearance. To assume that a periostitis was always the result of any one specific cause would be to contradict scientific proof. Hence to assume that the changes in the tubercle were always traumatic or always infectious would be to specifically exempt the tubercle from affections common to all osseous structures. The tubercle will react to either trauma or infection as will any structure in the bony frame and it is our contention that both injury and infection may produce the picture noted in these conditions. That trauma is the chief etiological factor is obvious but that infection may produce the same result, must be likewise obvious, from the cases we have studied.

INCIDENCE

We have found no reference to the frequency with which this condition is met. Osgood originally reported 7 cases and Boder 1 case in six years at the Cincinnati General Hospital. There have been numerous reports of several cases in the literature, but I am struck with the scarcity of articles in the

American literature. We have at our institution a series of 17 cases out of 16,000 general roentgenograms. Our figures represent an incidence of about 0.1% of all the plates taken and a total of 0.7% of all the knees examined. When one considers that many of the radiographs were taken of other parts of the body, many of them in adults, in whom this disease does not occur, one is justified in assuming that this condition is considerably more common than is generally believed.

The age series runs from 10 to 15. Most of the cases occurred between the ages of 13 and 14. One case is included, that of a man 31 years old, with typical appearance although not classified as an Osgood-Schlatter's disease.

Most of our cases (15 of the 17) occurred in males. Some writers have found this condition equally prevalent in the sexes. The frequency in the male is accounted for by the greater muscular development as well as the greater activity of this sex. In 5 cases in our series no history of injury was obtained after careful interrogation and of these two were definitely classed as of the infectious epiphysitis type. Of our series 9 cases showed involvement of the left side and 4 of the right side. An unusual finding was the occurrence of bilateral involvement in 4 cases.

SYMPTOMATOLOGY

The commonest symtom complained of is pain over the patellar tendon and pretibial tubercle. It is of somewhat variable character and may come immediately after or may follow months after the original injury. Often of so minor a character as to escape special notice, it is at times extremely severe and sufficient to cause marked disability. It is present at times as a local feeling of discomfort and often it occurs only on exercise of the part. It may be referred along the patellar tendon. It is intermittent in character and often recurs with increasing frequency until medical relief is sought. As a rule it is increased by motion and relieved by rest.

Limitation of motion, chiefly extension, is present as a rule. This symptom is also a variable one and depends on the existing damage to the part. In some the impairment of function is slight and is simply an inability to use the limb with comfort. In others the disability is marked, although no total loss of function was present in any of the cases in our series. Flexion is rarely interfered with, excepting where severe injury has resulted in marked periarticular swelling and synovitis.

Periarticular swelling is commonly present and is usually due to direct trauma to the part. Local contusions or even lacerations of the skin over the site of injury are the result of direct trauma. Pretibial swelling or protrusion is practically always present and in two of our cases this was practically the only condition for which relief was sought. While this latter is usually due to the separation of the tubercle it may at times be the result of the associated bursitis.

A reliable guide is local pretibial tenderness. In few of our cases was this absent. It can best be elicited on pressure over the tibia or rather the apophysis, with the limb in the semi-flexed position, so as to stretch the patellar tendon.

ANATOMICAL CONSIDERATIONS

A study of the anatomy and development of the structures under consideration will assist materially in the proper understanding of this type of affec-

Fig. 1.

tion. We must therefore consider the following:

1. The tubercle of the tibia.
2. The ligamentous attachments.
3. The relations of the bursae to this disease.

The tubercle of the tibia. The development of the tibia proceeds from four centers: one for the shaft, one for the lower epiphysis and two for the upper epiphysis. The centers for the upper end or head appear at birth and are united to the shaft between the ages of 21 and 25. The center for the lower end appears at about 2 and is united to the shaft between the ages of 18 and 19. The tubercle of the tibia develops from the tongue-like protusion of the lower and anterior aspect of the upper epiphysis. Often, however, the tubercle develops from two centers: one from the epiphysis of the head and one from the shaft proper. Our series include tubercles formed from three centers one from the shaft and two from the upper epiphysis. Two points from this study are then immediately evident; first, that while the center of ossification of the head appears first it is nevertheless the last to unite with the shaft and is not completely united at the age of greatest physical activity, and second, points of union are often present in the tubercle it-

self, permitting during the stages of development, of easy separation of the fragments[16].

2. *The ligamentous attachments.* The patella, the largest sesamoid bone in the body, is developed within the tendon of the quadriceps femoris, which is one of the largest, as well as strongest tendons in the human frame. By means of this tendon the patella is attached to the tubercle of the tibia. Aponeurotic fasiculi of the quadriceps form the patellar tendon and are continued as small strands from the tubercle to the lateral surfaces of the tibia at its upper end.

The patella is thus supported by, or suspended between dense fibrous bands and rides comparatively free between these two attachments. Nature has here employed an engineering principle. The point of interest in this connection is that the pier of the suspensory bridge is the tubercle and that the anchorage is further reinforced by the lateral restraining trusses or guy ropes of the terminal filaments of the patellar tendon attached to the tibial shaft proper.

The upper attachment of the patella is movable within the limits of contractility of the quadriceps femoris. The only fixed point at the knee so far as we are here concerned, is the tibial tubercle. The degree of motion in and about the knee joint will of necessity depend on the integrity of the pier head.

3. *The relations of the bursae of the knee.* These are:

1. Prepatellaris, subtendinea, between the tendon of the quadriceps and periosteum of the patella.
2. The three prepatellar bursae:
   a. Subcutaneous
   b. Subfascial
   c. Subtendinous.

There may be only two bursae instead of three and these connect laterally with the synovial membrane of the knee.

3. Suprapatellar, between the anterior surface of the femur and the posterior surface of the quadriceps.

4. Bursae between the patellar ligament and the anterior aspect of the head of the bone[17].

The close relationship between the bursae and the knee joint permits of the ready extension of the disease process to the synovial membrane. As a result there is often added to the picture of an avulsion fracture that of a bursitis or synovitis of the joint.

The affect of a sudden muscular strain on these parts is apparent from the anatomical relationships. The natural architecture of the joints is such as to safely withstand the usual trauma. Direct injuries, or sudden strains or stresses, particularly with the knee in the semi-flexed position, often results in partial or complete separation of the tubercle. The construction of the tubercle, together with its ligamentous reinforcements explains why, with severe traumas and complete separation of the tubercle, partial or complete extension at the knee joint is still possible. Severe injuries, even though they produce avulsion, rarely succeed in breaking the lateral struts; many of the fibers remain intact and extension is still possible through them.

ROENTGENOLOGICAL FINDINGS

For purposes of analytical study the roentgenological aspect of this condition will be considered from its effects on the bone, and on the soft parts. Although coexisting to a variable degree, they are nevertheless deserving of independent discussion.

Fig. 2.

Bone Changes. It is axiomatic that the degree of the force applied will determine the extent of the damage to the bony parts.

In mild muscular efforts very little or no injury, or at least a partial, incomplete, variable amount of separation of the apophysis results. Dunlop's cases of strain of the tubercle would come under this category. These cases may be negative radiologically or at least present a negligible or hardly discernible separation of the part. In these mild cases, the clinical symptoms are at times more marked than would be expected from the examination of the roentgenograms. We wish to emphasize that the clinical examination is as necessary here as is the investigation of any other type of pathologic process.

In direct trauma or severe muscular strains more extensive separation results. The amount of separation will depend on the degree of development of the part. In tubercles formed from the epiphysis of the head, the separation is, as a rule, *en masse*, and a definite space may be demonstrated between the tubercle and its osseous attachment. Figure 3

is of this type. In those formed from multiple centers of ossification the appearance presented is somewhat as follows: The distal fragment is the point of attachment of the patellar tendon and as such sustains the brunt of the injury. This segment is often pulled free and projects as a definite bony prominence on the anterior aspect of the knee.

Fig. 3.

Patients often will present themselves for this bony protrusion. In certain traumas not only does the tubercle itself become separated but often a portion of the anterior surface is torn completely from the tubercle proper and lies in the soft tissue anterior to it.

In the severe muscular strains the lower pole of the tubercle may be pulled loose and displaced upward and forward in front of its upper half and united in this position. Figure 3 is of this type. The types thus far enumerated may be classified as occurring from muscular strains, although direct injury may produce the same picture. Often, in direct injury, the picture of a comminuted fracture of the part may be produced.

We believe that this condition is much more common than is generally supposed, while failure to recognize it must be taken into consideration; still the absence of more marked symptoms might well prevent the average physician from seeing the cases when the injuries occur. We have already seen that some cases present themselves for cosmetic treatment rather than for the relief of pain. One must assume that many cases escape examination in view of the absence of acute symptoms. It is possible that sudden trauma may either activate a preexisting pathological status or else complete the separation in a tubercle that has been incompletely separated due to insufficiency of the forces applied. Figure 4 illustrates this well. Here, the history was of two days duration, yet on examination definite bone atrophy was present, proving that the condition existed in a somewhat quiescent form prior to the time of injury. That this condition may be present, without, in many cases, giving symptoms, is illustrated in figure 2, where there was a history of trauma three years before and yet the patient did not appear for examination until a trivial injury brought him under observation.

It is a medical truism that developmental structures are prone to infection, particularly cartilage and newly formed bone, be it in the spine, the head of the femur, or in the tibial tubercle. The frequency of osseous tuberculosis is an example. Cartilage, in particular, while so resistant to malignancy, is especially susceptible to infection. Low grade infections may involve the tibia or rather the tubercle, as an independent affection or as a local manifestation of a general disease. The exciting factor is often injury or a series of injuries of a minor character which in themselves may not produce symptoms but, if repeated, will produce a definite pathological entity. Reference to the anatomy already given explains how the local manifestation in the tibia may spread to and involve the knee joint.

When an infectious change involves the tubercle they present a slightly different appearance. In the early stages there is noted a fuzzy or somewhat irregular appearance or border. Following this acute stage reactive processes are noted. The substance as well as the margins of the bone become denser and small bony proliferations are observed extending outward from the periphery of the tubercle. These changes, although variable, are never extensive.

The result is what one commonly sees in any bone the seat of an non-suppurative inflammatory process. The cancellous structure becomes denser than its prototype of the opposite side. The cartilaginous limits disappear so that there is premature union of the apophysis to the shaft of the tibia, as is illustrated in figures 6 and 7. "Welding of the tubercle to the diaphysis may be prematurely produced by trouble in the epiphysis." (Lannelogue in the *Bulletin de la Société de Chirurgie*, 1878).

The attention of the reader is directed to a careful study of figures 6 and 7. The plates are of a 14-year old boy. Careful history failed to obtain any evidence of a previous injury or of a severe muscular strain on the part. The patient complained of pain only for three weeks prior to examination at the Hospital for Joint Diseases. With pain some pretibial swelling was present. The clinical examination showed the swelling complained of and a slight pretibial tenderness on deep pressure over

the anterior aspect of both knees. Roentgenography of the right knee showed the apophysis incompletely united to the shaft of the tibia. There was a dense sclerosis of the apophysis and a free fragment of bone was seen in the soft tissue in front of this process. This apophysis appeared to be formed from three centers; one from the shaft and two from the head of the bone. The changes on this side appeared to be the older of the two. The left knee showed the apophysis to be much larger than its fellow on the opposite side. The margins of this process were hazy and somewhat indistinct and a small exostosis was present on its anterior surface. This side appeared to be the seat of an active

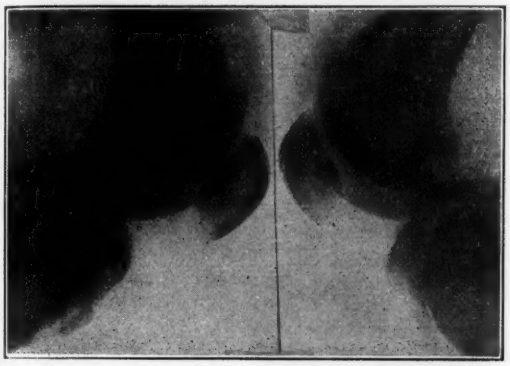

Fig. 4.

inflammatory process. There was a synovitis of both knees. It was apparent that we were dealing here with an old lesion, existing quiescently for three years, that suddenly gave rise to symptoms that warranted attention. This case cannot be classified as an avulsion of the pretibial tubercle, but belongs to the type of infectious non-specific epiphysitis of the tubercles.

Soft Tissue Alterations. The effects of the pathological changes occurring in and about the knees are in general terms the same. From whatever angle we view it, cause and effect are closely associated. The intimate relationship of the bursae and the synovial membrane, to which attention has been called, provides a perfect medium for the reception and transmission of inflammatory changes. The bursa overlying the tubercle is affected, even in mild cases, and in severe cases also the synovial membrane through the prepatellar bursae. Bursitis and synovitis then, are commonly present and radiographically demonstrated. Extravasation of blood, either as a subcutaneous hemorrhage or as a true hemarthrosis, occurs at times, but infrequently. We have seen it in only one case. Periarticular swelling is the result of both intra- and extra-articular injury, and is chiefly noted on the anterior aspect of the tibia and on the sides of the patellar tendon.

DIFFERENTIAL DIAGNOSIS

The changes in the main are typical, simple and readily recognized. The following conditions, however, may simulate this disease:

Sarcoma of the tibia
Tuberculous arthritis
Lues
Osteomyelitis

Sarcoma. The frequent occurrence of sarcoma at the upper end of the tibia is a fact commented on by most writers on the subject. However, sarcoma rarely involves the tubercle; we are not aware of a report in the literature. The absence of either

Fig. 5.

bone production in the soft tissue or of bone destruction in the cancellous structure, as a rule serves to differentiate the two. Sarcoma is not limited as is this condition. The history and clinical observation of improvement under observation is the rule in Osgood-Schlatter's disease, whereas in sarcoma, it is one of progressive involvement.

Tuberculosis of the knee. In severe injuries or infection the dense synovitis that results may be mistaken for an early involvement of the knee. In some cases, particularly in the infectious type, the cloudy appearance of the epiphysis may be mistaken for actual destruction of this part. However, the absence of intrinsic predominating joint involvement is the differential point. In tuberculosis, atrophy is a part of the disease and in the absence of mixed infection, invariably is present. In disease of the apophysis it is the result of disuse. No undermining of the joint surface results nor is there actual destruction of the joint cartilage in Osgood-Schlatter's disease.

Lues. The indistinct hazy appearance of the outline of the tubercle as well as the commonly associated synovitis may be mistaken for lues. The

predilection of congenital lues is for the chondro-periosteal margin at the epiphyseal end of the diaphysis and rarely for the tubercle proper. The lesions also differ materially in type. That of lues is, as a rule, of the gouged out or punched out areas and occasionally involvement of the periosteum (in adolescents). In Osgood-Schlatter's disease no gouged out areas occur, nor is there involvement of

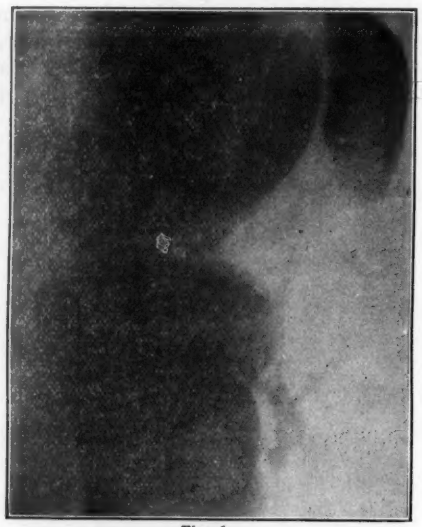

Fig. 6.

the periosteum. There is no atrophy in lues, as a rule, whereas atrophy from disuse sometimes occurs in Osgood-Schlatter's disease.

Osteomyelitis. In this condition the presence of bone production or destruction occurring simultaneously or independently, together with a periostitis will sufficiently differentiate this condition from Osgood-Schlatter's disease. Actual joint involvement in osteomyelitis of the long bones is uncommon and synovitis unusual. In marked cases the presence of sequestra is sufficient for a diagnosis.

We have attempted briefly, to differentiate this condition from allied affections. As a rule the history, careful clinical and radiographic examinations are in themselves sufficient to furnish an accurate and reliable diagnosis.

Our series consists of 17 cases: histories, physical findings and radiographs. Lack of space prevents a detailed description of all of these cases, but we have included a brief report of six that illustrate the subject matter that has formed the basis for this paper.

CASE No. 125330 (figure 1), Female, age 10. Reported to the hospital October 9, 1922. Clinical diagnosis; tuberculous gonitis. There was a history of injury at the age of 2, since which time patient has been unable to properly extend and flex the right knee. At times there was intermittent synovitis, pain and swelling. There were marked periarticular swelling, and a slight elevation of local temperature. There were also considerable tenderness on pressure over the knee, particularly anteriorly and some restriction of motion as well as muscular spasm.

Roentgen examination: Osgood-Schlatter's disease, right tibia, with marked bone atrophy. The tibial tubercle was pulled upward and forward and its lower pole was noted projecting under the skin surface. The atrophy present was due to disuse and not to intrinsic bone disease. No destructive changes were noted in the articular surfaces of the bones of the knee joint. The tubercle in this instance appeared to be formed from two centers; one from the shaft and one from the epiphysis.

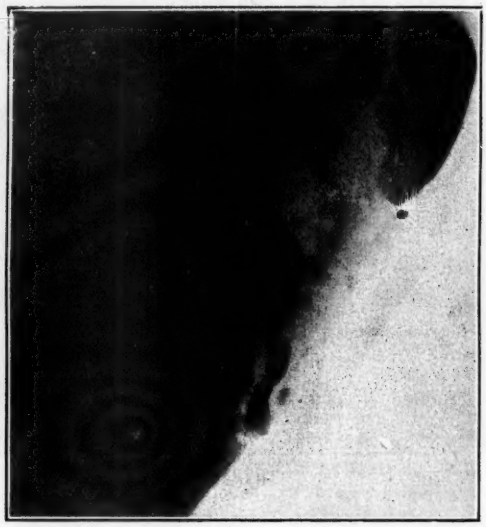

Fig. 7.

CASE No. 128225 (figure 2), Male, age 14. Reported to hospital January 1, 1923. Clinical diagnosis: Osgood-Schlatter's disease of the left tibia. Patient gave history of sustaining severe injury to the left knee about one month before observation at which time he was treated by family physician. Some relief was obtained but the pain and disability returned and a swelling over the anterior aspect of the knee became particularly painful.

Roentgen examination: Osgood-Schlatter's disease of the left tibia. The terminal segment of the tubercle was torn free from its attachment and displaced upward and forward and the lower sharp tip was noted projecting towards the skin surface. The tubercle appeared to be considerably denser than the subjacent bone. There was slight bone atrophy from disuse. Considerable pretibial swelling was present, but imperfectly reproduced on the print.

CASE No. 126851 (figure 3), Male, age 15. Reported to hospital November 20, 1922. Clinical diagnosis: old fracture, left tibia. Patient had slight injury to both knees several months prior to examination, but the injury was of so minor a character that no particular attention was paid to it at that time. A few days before examination some discomfort in the left knee, at which time he became aware of a pretibial protrusion, for which he sought attention.

Clinical examination: Both knees appeared to be normal, except for a slight protuberance on the anterior aspect of the left knee at the upper end. Some tenderness was present on deep pressure over the tubercle. The lower end had become detached and displaced upward and forward and united to the anterior aspect of the tongue-like process of the upper end. On close inspection of this roentgenogram, it was apparent that an osteoplastic change had resulted. There appeared to be some excess bone formation with a notable increase in the size of the tubercle proper In addi-

tion, an avulsion of the periosteum was noted at the upper anterior aspect of the apophysis.

CASE No. 108283 (figure 4) Male, Age 13. Reported to the hospital October 31, 1921. Clinical diagnosis: Osgood-Schlatter's disease, left tibia. History of injury to the left knee three years previously which did not in any way incapacitate him. Three days prior to examination patient struck knee against hard object since which time there had developed a hard painful swelling.

Clinical examination practically negative except for a tender prominent tubercle.

Roentgen examination: old Osgood-Schlatter's disease, left knee. This case was included here, because it demonstrated several important features. There was no history of injury prior to that which occurred three years previously. In this instance it was quite possible that there was an old avulsion of the tubercle which at the time was unknown to the patient. The repeated trauma, or rather the recent trauma and the roentgenographic examination demonstrated the tubercle to be completely torn from its attachment to the tibia. Close inspection of the plate demonstrated two shadows: one of the displaced fragment and the second that of the normally situated lower end of the tongue-like process of the upper epiphysis. It was noted that there was a synovitis, as well as some lipping of the articular margins of the bones of the knee joint, which indicated that there must have been a pre-existing arthritis.

CASE No. 127276 (figure 5) Male, age 12. Reported to the hospital December 12, 1922. Clinical diagnosis: synovitis of the left knee. No history of injury obtained. There was continuous pain in the left knee for two months preceding, associated with periarticular swelling and some disability.

Clinical examination revealed a distinct periarticular swelling and some limitation in the movements at the left knee. There was generalized tenderness in the soft structures about the knee.

Radiographic examination: Osgood-Schlatter's disease of the left knee. There were also present marked synovitis and periarticular swelling. The tubercle in this instance was pulled free from its tibial attachment and considerably displaced. The print does not demonstrate the free fragment clearly. Note the cortical thickening of the inferior angle of the patella.

CASE No. 131838. (figures 6 and 7) This case is described in detail in the text.

CONCLUSIONS

1. Avulsion of the pretibial tubercle occurs most frequently in the adolescent.

2. The disease is more common than is generally supposed. The absence of acute symptoms explains the scarcity of case reports.

3. The architecture of the joint is such as to safely withstand moderate trauma.

4. Most of the cases comprising this report occurred in males and in the left knee, which is at variance with the reports of other writers.

5. The lesions are most commonly traumatic, although two cases of the infectious type are here reported.

6. Dense reactive sclerosis occurs in old lesions.

7. The types of cases vary according to the severity of the trauma from simple strains or avulsions of the periosteum to complete avulsion of the tubercle with fragmentation of this process.

I am indebted to Dr. Henry W. Frauenthal for the use of the material of the department and to Dr. Herman B. Philips, roentgenologist of the hospital, for suggestions embodied in this report.

HOTEL ANSONIA.

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THE TREATMENT BY ASPIRATION AND MOBILIZATION OF TRAUMATIC JOINT EFFUSIONS ("TRAUMATIC SYNOVITIS")

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For several years it has been my practice to treat traumatic joint effusions—especially in the knee—by prompt aspiration and early movement. Aspiration in traumatic synovitis of the knee has been warmly advocated, and its safety and advantages have been demonstrated, by Willems[1], Harding[2], Moorhead[3], Metcalfe[4], McWilliams[5] (in the chronologic order named); and it was practiced by a few earlier surgeons. Yet it is to be observed that the time-honored, tedious, and often unsatisfactory treatment by prolonged immobilization and then prolonged massage, baking and douching, is still current in general practice and in the teachings of the text-books.

Whitman[6], however, concedes that "aspiration is indicated whenever the effusion is sufficient to cause tension or discomfort and should be repeated as often as indicated;" and one or two other text-books suggest that aspiration may be performed in cases

of great distension or prolonged swelling. The timidity of the suggestion conveys the intimation that the author regards the aspiration of a distended knee joint as a serious affair, not to be undertaken except as a resort to relieve great pain; and, indeed, many surgeons shrink from plunging a needle into a sterile distended joint for fear of introducing infection. In fact, however, aspirating the knee joint is more simple and more safe than aspirating the pleural cavity—yet the latter is an everyday procedure that is employed by practitioners generally. I have not seen infection or even reaction follow any of the many joint aspirations I have made; nor, presumably, has any of the authors cited, although one of them (Metcalfe) treated thus more than 300 cases.

Although one would elect to surround the field with sterile towels and to wear sterile gloves there is, in fact, no danger of infecting the joint if the skin is clean and iodinized and if the needle,—a long one, freshly and sufficiently "boiled"—is handled only by its butt. The only precaution worth especial mention is that each aspiration—if more than one emptying is necessary—should be through a fresh site. The knee lends itself well to the procedure. The puncture can be made equally easily at either side of the patella. It is difficult to completely empty this joint even with a good syringe; the process is facilitated by manual pressure, and by guarded active movements while the needle is *in situ*.

The ankle joint does not lend itself so well to aspiration. Moreover, in most cases of sprained ankle the swelling is chiefly an edema in the extra- and para-articular structures. However, I have twice aspirated an ankle joint, and once a finger joint, after trauma, with prompt relief.

In my experience the fluid removed early from a "sprained" joint is always bloody, and often, especially when the injury is by direct violence, it is practically pure blood—a hemarthrosis, rather than a hydrarthrosis. Even after several days the fluid shows traces of blood, both by its color and microscopically. Harding noted that "practically all [traumatic] effusions are bloody and absorb slowly;" and Metcalfe, who reported a much larger series, also found that "a traumatic knee aspirated a few hours after the injury invariably showed practically free blood, after 6 days a bloody serum, and after 10 days a straw-colored serum".

The text-book teaching that a sprained joint becomes distended with serum is therefore faulty. Clearly, too, we must alter our conception of the pathology of joint sprains. As concerns the knee certainly, and all other joints quite probably, there is in a "sprain" a tear of the synovial membrane, associated with some injury (break or tear) in ligament, cartilage or bone.

An infraction (crack) in the patella, femoral condyles or articular end of the tibia probably occurs more often in knee sprains than is recognized, for these injuries are not submitted uniformly to roentgenography (as they should be) and, moreover, a small fissure in the bone may escape detection in the *x*-ray films if the latter are not of good detail and carefully scrutinized.

I do not think that one can distinguish, by the appearance of the joint, between a blood and a bloody effusion. An external violence to the knee is apt to produce a hemarthrosis, but, to repeat, even the "sprain" of an internal violence is associated with more or less bleeding. It is no doubt because the blood absorbs very slowly that knee sprains take so many weeks to recover under the older plan of treatment (immobilization, massage, etc.)

The advantages of treatment of traumatic synovitis by prompt aspiration are many:—It relieves pain. It permits early or immediate mobilization, and thus prevents the atrophy that always develops in the immobilization treatment, and the stiffness that often develops in that treatment. It allows the distended joint capsule to contract and regain its tonus and thus minimizes the tendency to weakness that is a not uncommon sequel to the immobilization treatment. By emptying the joint it obviates, or reduces the likelihood of, the production of joint mice and synovial thickening by retained fibrin—a process that is believed by many authors to occur. It is a simple and inexpensive plan of treatment as compared with prolonged physiotherapy. Finally, and especially, it reduces the period of disability from many weeks to a few days, and the period of treatment from two months or more to two weeks or less!

The removal of the fluid from a sprained knee also makes possible the immediate resumption of walking. Perhaps in some cases, viz. those that come nearest to what one might consider a "simple strain" walking does no great harm; but I am quite satisfied that in fresh injuries it does no good. However, Willems, Moorhead and McWilliams all recommend *immediate* weight-bearing (walking) as part of the therapy. Both anatomical considerations and personal observations make me believe that they are wrong. To encourage early, if not immediate, mobilization of the sprained knee joint by active movement is one thing; to submit, unprotected, the stretched and perhaps torn capsule, the freshly traumatized, swollen and bleeding synovial membrane to the pressure, the strain and the torque of walking is quite another thing!*

Moorhead's inclination to immediate walking

*To be sure, we encourage immediate walking more or less regularly in ankle sprains, but here we are dealing with very different conditions: the ankle is a mortise joint and has no internal ligamentous or accessory cartilaginous structures; and to prepare it for weight-bearing we support, and more or less immobilize, it with adhesive plaster strapping. Moreover, the lesion in ankle sprains is largely extra-articular, as a rule.

after aspiration may have been the early enthusiasm of then limited experience, for in the last (second) edition of his treatise on Traumatic Surgery, which appeared the same year as his cited article (clinic), he described, as the treatment for acute synovitis of the knee, immobilization for two weeks or more, to be followed by massage; and he said: "It is rarely necessary to aspirate the fluid". In McWilliams' reported case, that of a traumatic hemarthrosis of the knee, he had the patient walking on the third day; the next (fourth) day the joint was again distended and two ounces of blood were withdrawn by a second aspiration; walking was continued, and the joint was emptied five times (!); the patient was pronounced well at the end of three weeks. I venture to assert that if this man had not been made to walk on the third day his knee would not have been found filled with blood again on the fourth day; and that if weight-bearing had been delayed for a week or so it would not have been necessary to aspirate five times and the period of treatment would have been cut in half. Contrast this case with Metcalfe's method. After emptying the knee joint he kept the extremity at rest in a traction apparatus for 9 or 10 days, and *then* allowed walking. His patients (soldiers) were restored to duty in 11 to 14 days; and he stated that in his more than 300 cases less than 25 required a second aspiration. (Incidentally, Metcalfe advises aspiration, not immediately, but after 48 hours, when the bleeding has ceased.)

Harding, too, is quite definite in his condemnation of forced motion and early weight-bearing, which he says is worse than the let-alone policy. I do not believe that immediate walking hastens the cure and I am quite satisfied that, by producing fresh bleeding, it often delays it. While early mobilization is desirable I believe this, too, should be restrained, more or less, for a day or two, until one can determine both the tendency to re-effusion and, as nearly as possible, the character of the injury.

Modified according to these last-mentioned considerations, I treat traumatic synovitis of the knee as follows: If the injury is recent the joint is emptied as completely as possible of the effused blood or bloody serum, and a compressive dressing is applied (gauze roller, or cotton wool and bandage), which allows but a slight range of motion. The patient is kept in bed or is allowed on crutches, without weight-bearing on the injured side. *X*-ray pictures are made in two or more directions and with instructions to secure bone detail in order to determine the presence of a fissure. The compressive dressing is discontinued after 24 or 48 hours, and free motion is allowed. If there is then fresh effusion in the joint, it is emptied by a second aspiration and the compression is (usually) reapplied. When there has been no reaccumulation for several days (a week or less, after the injury) the patient is allowed to bear weight on the knee—not walking freely as Moorhead advised, but guardedly, to test the tendency to fresh bleeding. If there is no return of swelling the walking is increased and after a few days of further observation the patient is discharged. If weight-bearing causes effusion, however, it is at once discontinued for another period of five to seven days, and the fluid is withdrawn with the needle.

If the injury is not a fresh one, immediate weight-bearing after aspiration is or is not permitted according as the fluid accumulation is moderate or large and contains little or much blood, and according to the character of the injury as determined by history, roentgenograms and physical examination. If the joint capsule has been much distended I think it unwise to permit unsupported weight-bearing until it has had a fair opportunity to contract.

The undesirability of immediate weight-bearing after aspiration is illustrated by two cases that were simultaneously on my service at the Broad Street Hospital.

Benjamin L., 31, was admitted November 29, 1924, within an hour after his right knee had been caught between two heavy rolls of paper. There were noted a slight laceration of the leg near the joint; tenderness over both medial and lateral heads of the tibia; and a considerable effusion into the joint and quadriceps bursa. Forty c.cm. of blood were withdrawn by aspiration, and the joint was bandaged over cotton wool. Roentgenograms showed no bone injury. Bleeding and coagulation times normal.

December 3rd: Moves knee freely. Some reaccumulation. Fifteen c.cm. of blood aspirated.

December 7th (ninth day): Allowed to walk.

December 8th: Joint found again much distended. Sixty c.cm. of blood evacuated. Put back to bed for one week.

December 15th: Again allowed to walk.

December 18th: No reaccumulation of fluid. No pain or discomfort. Discharged, well.

Emerson C., 18, admitted December 3, 1924, had suffered the first trauma to his left knee six months previously when the medial head of the tibia was struck by a falling anchor. The joint became distended, but the patient continued to walk and sought no treatment. Thereafter the swelling never entirely disappeared, but varied in amount, and the knee sometimes felt weak. Two weeks before admission the left knee was again struck on its medial aspect by a packing case. Great swelling followed, but the lad continued to walk.

On admission there was a painless, massive effusion in the joint and quadriceps bursa, and an abrasion over the medial head of the tibia. Two hundred c.cm. of yellow-pink serum were withdrawn by aspiration. No bandage was applied, and free movement of the joint was allowed, but the patient was kept in bed because roentgenograms showed typical osteochondritis dissecans and lipping of the medial condyle of the femur (figure 1).

This lesion is rather generally regarded as traumatic in origin; and, indeed, Arthur Krida[7], has recently described it as "osteochondral fracture of the knee joint"—an early phase of König's osteochondritis dissecans. It occurs typically in young adults from a blow on the knee, and consists of a separation, without immediate displacement, of a segment

of the articular surface of the medial condyle of the femur.

In this case there was no reaccumulation of the effusion after the aspiration. To avoid, if possible, displacement of the osteo-chondral fragment, the patient was kept in bed until December 23rd and then discharged on crutches, without weight-bearing on the affected side.

A roentgenogram two weeks later showed the fragment still in its bed but exhibiting evidence of still further separation. There was then slight effusion. Upon trial of weight-bearing massive effusion took place. Accordingly, on January 12, 1925, through a split-patella arthrotomy (at Broad Street Hospital) I exposed to view the uniformly swollen, deeply injected synovial membrane, and a swollen synovial fold (fringe) over the "lipped" margin of the medial condyle; and I removed the adherent but ununited osteo-chondral fragment and *also a much larger, (2 x 4 cm.), immediately adjacent and rather less adherent fragment of condylar cartilage, the formation of which had*

Fig. 1. Osteochondritis dissecans (osteochondral fracture of the medial condyle of the femur) in a case of recurring traumatic synovitis of the knee.

not been indicated in the roentgenograms and probably would not have been demonstrated by pneumoroentgenography. (figure 2).

It seems quitely likely that such a cartilage fragmentation which, as contrasted with osteo-cartilaginous fragmentation, may be demonstrable only by open operation, may often be the cause of recurrent (traumatic) synovitis, not otherwise explained.

SUMMARY

The effusion in a joint sprain is not clear serum, but is bloody. Often in traumatic synovitis, especially by external violence, the distention is by pure blood. Such a hemarthrosis is not distinguishable, by appearance, from a hydrarthrosis.

In acute traumatic synovitis there must be a tear of the synovial membrane and some injury to ligament, bone or cartilage.

All cases should be submitted to roentgenography; it may disclose a fissure-fracture of tibia, femur or patella.

A cartilage fracture, as on a femoral condyle, not demonstrable roentgenographically, may be the cause of a chronic traumatic synovitis with persistent or recurrent effusions.

The time-honored treatment of synovitis of the knee by immobilization, massage, etc., is tedious and may leave a weakened joint.

Treatment by prompt aspiration of the blood or bloody fluid, and by early mobilization, is safe and simple, and shortens the period of disability by several weeks. It relieves pain, obviates atrophy, removes or minimizes what danger of further trouble may be inherent in retained fibrin, and leaves a stronger joint.

In acute cases actual weight-bearing *immediately* after emptying the joint is not recommended. It encourages bleeding and delays rather than accelerates cure. It should be avoided for about a week, meanwhile permitting movement of the joint in bed or on crutches.

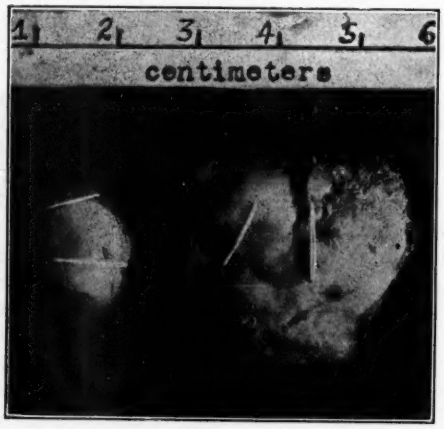

Fig. 2. Large chondral and small osteo-chondral fragment removed from the medial condyle in the same case as figure 1.

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THE TREATMENT OF ACUTE TRAUMATIC SYNOVITIS*

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The incubus of conservatism, and the weight of authority have ordained in the past that synovitis be treated by rest, cold and later hot applications, by gentle massage and then by only very careful resumption of function. Whether this therapy is a result of the terrible consequences of joint-meddling in the pre-aseptic period of surgery or not, is immaterial. The upshot of the matter is that the traumatic, as in fact all synovitides, are for the most part treated by rule of thumb without any adequate conception of either the underlying pathologic or the subsequent healing processes. The result is that patients are wholly or partially incapacitated for a period of from three to six weeks and that the affected joint may be permanently weakened and particularly susceptible to subsequent injury and the formation of chronic exudates.

From time to time, some of the great masters of surgery have ventured into the untrodden paths Ambrose Paré was among the first to suggest the opening of joints and some forty or more years ago Lucas Championnière advocated mobilization of traumatized joints. The circumstances were unpropitious, or the times unripe and their leadership was forsaken. As early as 1887, however, Bondesen[1] brought evidence that the conservative treatment of traumatized joints was vastly inferior to the so-called more "radical" treatment. In Copenhagen, the puncture of hemarthroses had apparently been common practise for many years. Bondesen reported a series of 119 cases observed over a period of 20 years and showed that in 57 cases routinely punctured, 86% were cured completely in an average of 21.3 days, while in 62 cases not punctured only 62.9% were discharged cured after an average of 37.5 days. In 1897, Düms[4], a German army physician observed that since he had made it his practice to incise extensive hemarthroses in soldiers he had seldom seen the frequent recurrence of exudation previously observed, even during the most severe marches. His patients, observed during the whole period of their enlistment, remained completely free of any symptoms.

Gradually voices were raised in favor of more radical treatment. In 1905 Flint[6] wrote that in his opinion, "Every traumatized knee showing localized tenderness and effusion into the knee joint should be aspirated, provided there is not a marked diminution of the effusion under treatment at the end of one week". Later Rochard in a communication to the Société de Chirurgie de Paris advocated early puncture and passive motion against progressively increasing resistence. Willems[17 18] followed and amplified Rochard's treatment by permitting his patients to walk. "I commenced with evacuatory punctures to drain traumatic effusions of the knee, hemarthroses and hydrarthroses, and by making the patient walk immediately. Not only could they do this without difficulty but their lesions cured in a few days without leaving any trace". His cases were discharged cured in from 6-16 days. Since then other accomplished clinicians have advocated similar treatment for traumatic synovitides. Thus McWilliams[11] states that the most efficient method of treatment "appears to be the combination of repeated aspirations with immediate mobilization (walking and active movements)." Moorhead[13] states that "the best procedure after aspiration is to make the patient walk and use the knee in as nearly a normal manner as possible." This "limits the possibility of stretching of the synovial capsule and the parts contiguous and prevents in a large measure organized exudate which is a very potential source of loose bodies." Even Jones[10] in 1916 wrote, "after injury to the knee, effusion of fluid into the joint is very liable to occur and if the patient is kept resting and not using the limb, effusion will probably recur the first time he uses it. The rational treatment in such cases is to bandage the knee firmly and let him walk". Still the great majority of surgeons of the world adhere to the orthodox conservative treatment of yesterday and only a small minority grudgingly advises the aspiration of large effusions.

In the out-patient department of the Hospital for Joint Diseases, which draws its clinical material from the poorer laboring classes, the radical type of treatment outlined, became almost inevitable. The laborer, dependent on his daily toil for his livelihood, becomes an economic loss to society and a financial drag on his family, when incapacitated. Any procedure that can lessen the period of his convalescence or permit his continuing at work during his convalescence, is of immediate interest to society and of vital significance to him. In an army, during conflict, such a procedure, by releasing hospital beds and rehabilitating incapacitated combatants, would be of importance. The results obtained in our dispensary have been so satisfactory, that though the series is small, I have felt justified in presenting them. The cases here reported comprise only those in which there was a definite history of recent trauma with signs of effusion into the joint. Synovitides apparently due to infectious causes were as much as possible excluded. All but one of the cases consisted of injuries to the knee joint. In none was there evidence roentgenologically of any fracture, nor was there clinically any abnormality of motion as evidence of a gross ligamentous injury. All were repeatedly aspirated routinely, and

*This study was undertaken at the suggestion of Dr. Walter M. Brickner, Attending Surgeon, Hospital for Joint Diseases, to whom I am indebted for the privilege of publishing it independently.

all were enjoined to continue their normal pursuits. It was believed that by repeatedly aspirating the effusion there would be no opportunity for the development of a relaxed capsule, which leads ultimately to a weakened joint. It was further believed that by removing the fibrin-containing fluid, the chances of leaving a small fibrin mass which might act as the nucleus of a subsequent loose body would be minimized. Finally it was believed that by having these patients go actively about their usual pursuits there would be a greater tendency for early absorption of the fluid and a substantial diminution of the danger of quadriceps muscle atrophy with consequent tendency toward weakening of the joint.

Necessarily, the procedure employed has not been complicated. On admission, an adequate history is taken, a physical examination is made and a roentgenogram is ordered. Then, using only the usual measures that a surgical conscience prescribes, the joint is aspirated. An ordinary syringe, armed preferably with a 19 gauge needle, is used, and the joint is punctured through the iodinized skin area. The knee was usually punctured through an area a little to the inner edge of the patella and about an inch from its lower border, but it may be punctured with equal facility from the outer side. In the case of the wrist, the needle was simply inserted into the tense synovial sac. Care should be taken not to injure the cartilage or synovial membrane, else a fresh hemorrhage into the joint may be occasioned. Even in children no anesthetic need be used. There is a certain amount of pain, but not so much that a sensible child of six cannot stand it. Following the aspiration, the puncture wound is sealed with a bit of sterile adhesive tape and the patient is ordered to use the joint immediately. On the second day following, he returns to the clinic. If the fluid has reaccumulated in greater amount, the indication would be to discontinue the weight-bearing. This did not occur in any of the cases seen; and consequently the process of tapping the joint was continued as long and as often as any fluid could be obtained. Ordinarily three or four tappings were quite sufficient to completely free a joint of its fluid. I do not feel that the aspiration alone is responsible for this; doubtless the massage attendant on a functioning musculature plays a large part in bringing about an absorption of the effusion. In some of the cases baking and massage were prescribed, in others not. These measures are desirable adjuvants, but are no more essential in the outline of treatment than is the wearing of an elastic bandage. The results have been uniform. In a period of time varying from 6 days to about 3 weeks the patients, ambulant throughout, were discharged cured. Functionally, these patients have had no complaints, and no interference with point motion, and in some of the cases even anatomically, as far as could be judged clinically, the parts have been restored to normal. Thus in the case of the knee joint the swellings of the inflamed alar ligaments, so constantly seen along either side of the patellar tendon, recede, and there remains little if any clinically demonstrable peri- or intra-articular swelling.

CASE I.—H., 21, stock-clerk, was seen May 15, 1924, with a history of having on the previous day jumped over a case and twisted the left knee. He was able to walk, but with a definite limp and was unable to completely extend the leg upon the thigh without marked pain. There was a definite point of tenderness over the left internal lateral ligament with a moderate boggy swelling on either side of the patellar tendon. There was definite patellar ballottement, but no abnormal motion nor obstruction to motion in any direction. The *x*-ray picture showed a dense synovitis, but no fracture. On the 14th, 20 c.c. of a viscid bloody fluid were aspirated. Microscopically this fluid contained a few epithelial cells, many rouleaux of red blood cells, but no fibrin. Immediately following aspiration, the pain disappeared and the patient was able to extend and flex the knee fully. He was ordered to work. On the 16th, 25 c.c. of a slightly less bloody fluid were aspirated. A small number of fine fibrin shreds were seen floating about in the fluid which was slightly thicker than at the previous aspiration. On the 19th the fluid at first removed was straw colored, and syrupy in consistency. On standing, two large fibrin masses settled to the bottom of the test tube. At the end of the aspiration the needle scratched the surface of the patella and a few drops of bright red blood were aspirated. No significance was attached to this until the patient's next appearance at the dispensary, when it was discovered that the effusion had recurred and was again bloody in character. By the 27th the fluid had again completely disappeared, and the patient was discharged. During this whole time he complained of no pain and attended to his work. At the end of the month he went to the Plattsburg training camp with the strict promise to return for observation should he experience any inconvenience in his knee. He has not since been heard from.

CASE II.—W., 22, druggist, was first seen on May 21, 1924, with a history of having two days previously twisted his leg in jumping onto a case: He felt what he said was a "crunching of bone" and complained of pain in the suprapatellar region, which later localized to the region of the internal lateral ligament. He was able to stand, but was unable to completely bend his knee and complained of a feeling of insecurity in walking upstairs. The patient gave no history of locking nor of any previous joint trouble. There was definite effusion, swelling of the knee on either side of the patellar ligament and slight tenderness over the internal lateral ligament. No abnormal mobility could be noted, and there was no mechanical obstruction to passive motion. The *x*-ray report was "a dense synovitis, right knee with hypertrophic bone spicules at the margins of the articular surfaces". On the 21st, 15 c.c. of bloody, syrupy fluid, which partially clotted on standing, were aspirated. The pain disappeared and the patient was able to move his knee more freely. On the 23rd, 20 c.c. of a less bloody fluid were aspirated. On the 25th, 15 c.c. of only slightly blood-tinged fluid were aspirated. The patient still complained of a slight sense of insecurity in climbing stairs. On the 27th, 15 c.c. straw colored syrupy fluid were aspirated. On the 30th, the fulness about the patellar tendon still persisted, but aspiration revealed no fluid. This patient wore a knee cap and had several bakings, but did not miss one day from his work. He was seen again in December, 1924. During the whole time he had had absolutely no trouble in going about his usual work. A slight swelling about the patellar tendon still existed.

CASE III.—Miss R., seen August 4, 1924, had fallen on her left hand the previous evening. She complained of pain and inability to use her wrist. A moderately tense, painful swelling over the left radio-carpal joint was seen. Extension of the wrist was attended by pain. *X*-ray examination showed an atrophic arthritis, but no evidence of fracture.

About 3 c.c. of pure blood were removed from the joint with immediate relief of pain. When next seen, the patient could move her wrist normally. She was given massage and baking and has had no trouble since.

CASE IV.—E., 17, school boy, was seen on September 17, 1924. Two weeks previously he had fallen and twisted his knee. The knee became greatly swollen, but the pain was not excessive. An *x*-ray picture taken at Fordham Hospital was reported negative. He was treated by his family physician by the application of ice bags and a posterior splint. After 2 weeks of this treatment, without any improvement, the patient was referred to the Hospital for Joint Diseases. There was definite patellar ballottement and inability to flex the knee. No locking or abnormal mobility was observed. There was a moderate amount of fluid in the joint. About 15 c.c. of dark brownish-red, syrupy fluid were removed. Doubtless a great deal more was present, but could not be aspirated. The splint was immediately discarded and the patient ordered to move his knee. At first it was somewhat stiff, but after a short time he was able to bend it sufficiently to mount a chair, using the injured foot first. On the second succeeding day he was again aspirated, and two days later the fluid was quite clear, of straw color and slightly viscid. Following this his knee was baked and massaged, and on October 12th he was discharged with only a slight swelling on either side of patella, but with complete joint motion.

CASE V.—R., female, 6, was first seen December 10, 1924. While playing in bed two days previously she had sat down upon her bended leg. The knee swelled and she complained of slight pain. There were no tender areas about the knee, and there was no abnormality of motion. The *x*-ray examination showed only a "synovitis," but no evidence of fracture. The patella was floating and on aspiration 20 c.c. of a slightly viscid fluid, almost entirely blood, were removed. There was slight swelling on either side of the patella tendon. The patient was sent home and permitted to play about. On the 12th she returned, saying that she had been skating and playing with other children. She was again aspirated, but only 5 c.c. of the same bloody fluid as at first were obtained. On the 15th no fluid was thought to be in the joint, and the child was sent for baking and massage. The swellings alongside the patellar tendon had completely disappeared. The child was seen some three weeks later. She had been running about with her playmates and had not complained of any trouble.

Several other cases, some as old as two weeks, were aspirated and in all blood was found. Careful inquiry was made as to the existence of any tendency toward hemophilia either in the patient or in his immediate family. In none could such a history be elicited and so more elaborate procedures for determining the character of the blood, or the condition of the vessels were not employed. None of our cases showed any abnormal range of motion and in none did the *x*-ray show any bony solution of continuity. Indeed, had we seen any such cases we should have been tempted to exclude them from the category of simple traumatic synovitis. Had any complication such as avulsed tibial spine or torn ligament existed, the diagnosis and the consequent treatment would have been conditioned by this complication rather than by the presence of the bloody effusion.

One of the most striking consequences of our routine aspirations of traumatized joints was the establishment of the fact that all showed fluid blood in the joint cavity. It was only after the blood, apparently effused at the time of injury, had been removed that the fluid took on the serous character considered typical of "water on the joint". Though numerous authors have indicated the possible occurrence of blood in a traumatized joint, the frequency, one might almost say the regularity, with which a bloody exudate is found, has been but little appreciated. Harding[8] reports that in seventy cases of acute knee sprain seen at Camp Lewis, all had effusions. Of these, forty-six were aspirated, and eighty-seven percent showed pure blood, or a blood-tinged synovia, varying in amount from thirty to one hundred cc.; of the others about half had been known to have shown blood at earlier aspiration. Metcalfe[12] reports that in one hundred and fifty cases "traumatic knees aspirated a few hours after injury invariably show practically free blood, after six days of bloody serum and after ten days a straw-colored serum." Cooperman[5] reporting a large series of injuries to the ankle joint states, "usually there is a bloody exudate into the joint, its quantity depending on the degree of violence". I feel that a bloody effusion is not merely a possible contingency in a recent, acutely traumatized joint, but a constant occurrence and, in fact, a part of the clinical picture. That this conception has not gained wider credence in medical circles can be explained on the ground that aspiration has not been consistently nor sufficiently early undertaken.

When such acutely traumatized joints are seen on the operating table, the synovial membrane appears as a soft, red, swollen, succulent tissue which the earlier French writers recognized and described as a "pachysynovite". The sub-synovial vessels of the "circulous articuli vasculosus" are dilated and congested and there is an apparent new formation of vessels. Small fibrin shreds are to be seen adherent to the synovial membrane; the post-patellar pad and the synovial villi are congested, thickened and hypertrophied; the capsule is stretched and relaxed; and the synovial bands, (ligamentum mucosum and ligamenta alaria) may be thickened or torn across[6]. Microscopically, the synovial tissue is seen to be infiltrated by numerous small round cells and polymorphonuclear leucocytes, and there is some proliferation of the fixed tissue cells and of the endothelial lining of the joints.

This is hardly the place to attempt, nor do I believe does the state of our knowledge warrant, a complete explanation of the physiology of traumatized joints. Certain observations, however, throw interesting side-lights on the question and are of value as indicating the rationale of treatment. Normally the synovial membrane consists of a layer of flattened epithelioid cells which covers the interior of the joint everywhere except on the weight-bearing surfaces of the cartilage. The synovial cells are held together by a small amount of intercellular substance, but send anastomosing processes into the

connective tissue upon which they are supported. The lymphatic sinuses and the vascular network, in a word, the usual absorbing mechanism, lies in the subsynovial tissue, covered by a layer of epithelioid cells, and never comes into direct contact with the interior of the joint. This anatomical fact, taken in conjunction with the fibrosis of the subsynovial tissue, is of tremendous significance in explaining the length of time an exudate may remain in a joint. One of the cases here reported showed fluid blood almost three weeks after the initial injury.

Fluids are absorbed from joint surfaces by different mechanisms, depending apparently on the size of the particles of the fluid. Watery substances, as Braun[2] showed, are quickly and easily diffused through the intercellular substance. Suspensions of granular dyes were disposed of in different manners, depending on the size of the granules. The smallest sized granules diffused into the intercellular tissues and were thence conveyed to the lymphatic channels. Somewhat larger granules were taken up by the neighboring connective tissue cells and leucocytes. Still larger granules were taken up directly by the cells of the synovial endothelium, and a certain number, the largest granules, remained in the joint and were covered by fibrin masses and later by proliferating synovial cells. Jaffe[9] working with joints into which whole blood had been injected, showed a somewhat similar series of circumstances. Immediately after the injection, the synovial membrane became markedly edematous and injected as a result of the "permeation of the blood into the synovial tissue, in consequence of the marked intraarticular pressure". Adhering to the synovial membrane were fibrin masses in which organization was beginning and over which the endothelial cells had already commenced to proliferate. The synovial villi were thickened and hypertrophied, and there was a marked congestion and proliferation of the subsynovial articular vessels. Microscopically, the synovial membrane showed a cellular infiltration consisting mainly of small round cells focally arranged. This is exactly the picture found in acutely traumatized joints and the presumption may justly be made that the process of absorption of an effusion is analogous to the process above described. Occasionally, as Volkmann[16] repeatedly observed in his clinical lectures, the effusion may escape through a capsular rent and thence be rapidly absorbed by the surrounding tissues.

To both the physician and the patient, the appearance of "water" in the joint is the most striking phenomenon. Its onset is dramatic, its disappearance tardy, and its persistence fraught with the most serious sequelae. Immediately following the appearance of the effusion, various joint structures are placed under marked tension. The capsule is stretched and the stability of the joint is impaired. The torn synovial bands are pulled apart and normal healing is prevented. To compensate as much as possible for this increase in the intra-articular pressure, the joint adapts itself to the position in which its fluid capacity is greatest. The muscles, being mechanically splinted by the excess of fluid in the joint and consequently being deprived of the normal proprio-ceptive impulses from the joint, begin to atrophy. Payr[14] observes that in "inflammation of joints, atrophy, a reflex atrophy, of the neighboring muscles, especially the extensors, develops early." Garré and Borchard[7] insist on the necessity of energetically combatting the "muscle-atrophy by means of systematically progressive active motion". That this fear of early atrophy of muscles, in the case of the knee joint of the quadriceps especially, is not unfounded, is evidenced by the general attitude present-day surgeons assume towards the early mobilization of joints and muscles, even in cases of fracture. If in addition it be accepted that the mere presence of blood in joints predisposes to stiffness, the development of joint adhesions, and the precipitation of fibrin masses, which may act as the nuclei of joint mice, the urgent necessity of early evacuation of the fluid becomes apparent.

Treated in the usual manner, many of these patients are subject to frequent recurrences. Many are in later years afflicted with Hoffa's disease, a condition characterized by inflammatory hyperplasia of the ligamentum mucosum and the ligamenta alaria[7]. This disease is traumatic in origin and may be looked upon as the chronic condition of the pathological processes found in the acutely traumatized knee joint. Others become sufferers with chronic arthritis. In the opinion of Smith[15] both conditions, the villous hypertrophic changes and the changes of chronic arthritis are to be considered as traumatic in origin. He states, and quotes Wollenburg in the opinion, that "the two conditions are part and parcel of the same disease and that both are produced by chronic vascular changes, the causative factor being irritation of toxic or traumatic origin".

Whether the aspiration treatment will prevent the development of these late sequelae or not, cannot be stated at this time, but the procedure is rational and meets certain definite indications. By evacuating the bloody fluid promptly and repeatedly, the distention and the consequent laxity of the capsule is prevented; the separation of the torn ligaments and the tendency to their repeated trauma, and thus possibly to the later sequelae is avoided; the precipitation of fibrin masses and the possible consequent formation of joint mice and adhesions is minimized. By beginning active motion early, atrophy of the muscles is combatted and the absorption of the exudate is favored.

All of our cases have been permitted, indeed, ordered at once to go normally about their tasks. Brickner and other careful observers, surgeons whose opinions are to be given most thoughtful consideration, have maintained that such a procedure was unphysiological and unnecessary in that the same results may be obtained without permitting *immediate* weight-bearing with its possibility of fresh joint injury. It is not my intention to be dogmatic in insisting on weight-bearing at all costs and under all hazards. I have merely felt with Willems that "the goal to be reached is to restore the physiological function of articulation as much as possible, and in the case of the knee, this function is walking". That this contention is not wrong, at least in a certain number of cases, is evidenced by the results obtained. In none of the cases here reported was there any inconvenience or any symptoms of renewed injury. Had there been any, it would most probably have been manifested by the reappearance of pain and the reaccumulation of fluid. Naturally the only course open in such a contingency would have been to interdict weight-bearing while still permitting active motion of the joint.

After all, the physician, considered as a social factor, must view his responsibilities in a social light, and his patients in their social environment. I feel that if the patient, who would otherwise become a financial burden to his family, can be treated in a manner that will permit him to earn his livelihood without causing more than a slight increase in the length of his convalescence, the social gain is of such importance as to weigh the balance strongly in favor of the ambulant form of treatment.

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SYPHILITIC ARTHRITIS, WITH PARTICULAR REFERENCE TO SOME NEW PHASES OF ROENTGEN DIAGNOSIS

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It is generally conceded that joint syphilis is frequently overlooked or mistaken for other arthritic conditions, although less now than previously, by reason of more routine roentgen examinations and better recognition of characteristic roentgen appearances. Frequent reports of unrecognized luetic arthritis treated over long periods as tuberculous or other conditions appear both in foreign and domestic medical literature.

Henry W. Frauenthal[1] in 1906 called attention to the gravity as well as the frequency of mistaken diagnosis in this condition. Broca[2] claims that nineteen out of every twenty cases of syphilitic arthritis have persisted for months with alternating periods of improvement and aggravation, where the lesions are almost entirely indolent and do not interfere much with the duties of ordinary life. One would expect the percentage of errors to fall perceptibly, with the introduction of the Wassermann test, but the contrary is the case, for the errors in diagnosis seem more frequent now than before the advent of the Wassermann reaction. This is readily attributable to the credence given the Wassermann reaction in bone and joint disease, which appears distinctly unwarranted in the experience of the author and his associates. Roberts[3] states that the Wassermann test is not wholly dependable, and that negative Wassermann reactions are not sufficient evidence to exclude syphilis. Fisher[4] stated before the Orthopedic Section of the American Medical Association in 1917, that a considerable number of syphilitic bone and joint conditions simulate tuberculosis and that a considerable number, in his experience about ten percent., of syphilitic cases do not give positive Wassermann reactions. In the discussion following his presentation, all the speakers concurred in all respects with the points made, some maintaining that as high as fifty percent. of positive syphilitic bone and joint diseases give negative Wassermann reactions. Inasmuch as some of the most eminent orthopedists in the medical profession participated in the discussion and their opnions are so uniform, their names are quoted in the order of their discussion: H. W. Frauenthal, John Ridlon, Jos. Watkins, Edward Hatch, R. H. Sayre, C. B. Francisco, R. W. Lovett, E. S. Geist, and A. E. Horowitz. Nichols[5] states that fifty percent. of cases of latent bone syphilis give negative Wassermann reactions and that we should rely rather on the *x*-ray, than on any other factor for diagnosis. Poehlmann[6] is convinced that joint syphilis is common, but is usually mistaken for other affections. The Wassermann re-

action is often negative with syphilitic disease of bones and joints. It may be negative even in the puncture fluid from the diseased joints in known syphilis. The normal synovia, however, allows the passage into the joints of the substances giving the Wassermann reaction and also of stains and of bile pigments in jaundice. The inability to differentiate syphilitic joint disease fully justifies tentative treatment for syphilis in cases of obscure joint disease. *Notwithstanding this uniform opinion ex-*

Figure 1. Syphilitic arthritis discovered in course of roentgen examination for fracture. Longitudinal fracture can be seen about three quarters of an inch from external margin of external articular surface, of the upper end of the tibia. The absence of bone atrophy, commensurate with the duration of the fracture, aroused suspicions of syphilis, notwithstanding the lack of focal periostitic lesions. Reexamination with lateral views at varying angles brought out the characteristic focal periostitis which in the original roentgenograms had been covered by the patella.

pressed by representative orthopedists throughout America and the continent, diagnoses of luetic joint conditions in the face of negative Wassermann reactions are subject to criticism and even rejection, except in orthopedic circles.

In the present consideration of one hundred cases of syphilitic joint conditions, the Wassermann reaction was taken routinely in all cases, but it was not permitted to be controlling in the diagnosis. The ultimate deciding factor in establishing the nature of the pathologic process was the therapeutic test. Early experiences with this condition were such that very considerable reliance was soon placed on characteristic roentgen appearances. Negative Wassermann reactions were considered of no value in the face of positive characteristic roentgen findings. This is quite at variance with Jones and Lovett[7], who maintain that the Wassermann reaction runs parallel with the clinical and pathological findings in almost every case. In addition, these authors claim that the *x*-ray is of no value in making a diagnosis at any stage of a syphilitic arthritis or synovitis.

It must be constantly borne in mind that the *x*-ray offers a method of examination and diagnosis second in value only to microscopic tissue examination. The exact gross living pathology is projected onto the radiographic film. Projections in different planes and from different angles, with varying technics, afford exact and unvarying records of the disease process. The indispensable additional factor is, of course, a competent interpretation, which is just as

Figure 2. Comminuted fracture, tibia and fibula. Syphilis unsuspected. Symptomless prior to fracture.

necessary as in other fields of specialism. One should not expect a surgeon or general practitioner who may devote less than five per cent of his time to roentgenological considerations to do justice to such highly specialized work, any more than one could trust such a practitioner with the responsibility of diagnosing a microscopic section of a tumor. This flagrant disregard or lack of appreciation of characteristic roentgenographic appearances, is keenly appreciated by W. M. Brickner[8] who, in an article on the "Differential Diagnosis of Syphilis and Other Bone Diseases" states: "That surgeons and even many radiographists, so often fail to recognize the skiagraphic features of bone syphilis is not surprising when we find that several *x*-ray text-books describe them insufficiently, and that most surgical works say little or nothing concerning them. In Bevan's translation of Lexer's 'General Surgery,' published in 1908, the paragraph on diagnosis of syphilis of the bone, dismisses the *x*-ray aspect with the sentence, 'Roentgen ray pictures reveal nothing characteristic,' which statement is quite the reverse of the truth. The radiographic features of bone syphilis

are so characteristic that in most cases the diagnosis can be made from the *x*-ray plate alone, without or in the face of, clinical appearances and blood reaction. The exceptions to this rule are probably not more numerous than the exceptions to many other diagnostic signs and reactions."

This consideration of 100 syphilitic joint conditions is part of an analysis of a larger series of 200 patients with syphilitic bone or joint affections. Approximately thirty-five percent. of all had syphilitic arthritis (or synovitis), seventy-five percent. had

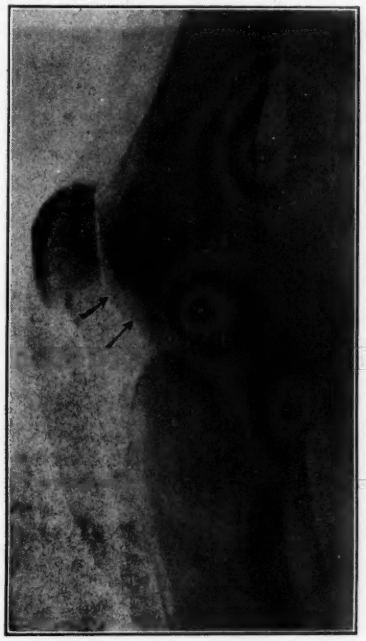

Figure 3. Syphilitic arthritis—focal periostitis, internal condyle anteriorly at chondro-periosteal junction. Atrophy not commensurate with duration of condition, two years. Further examination showed diffuse syphilitic ostitis of cranial bones and syphilitic osteomyelitis of the femur.

bone lesions, and ten percent. had both bone and joint lesions. Eight percent. of the patients had Charcot joints, of which six had multiple and ten single joint involvements. Forty percent. of the cases of syphilitic arthritis were of the knee joint and fifteen percent. of the elbow joint and spine involvement was present in three percent. Six cases were complicated by other infections or severe trauma. Multiple joint lesions were present in a great majority of the congenital cases.

Two cases of syphilitic arthritis were discovered in the course of roentgen examinations for fractures of the adjoining bones. After the roentgen diagnosis had been made, questioning brought out the facts that fluctuating swelling had been present in the joints for several months, with very slight (practically negligible) disability. This characteristic of lues of the osseous system, has been forcibly impressed upon my mind by the accidental discovery, by roentgen examination, of no less than nine cases of luetic osteo-periostitis, and of two cases of luetic arthritis. All of these cases were referred to the *x*-ray laboratory for examinations of fractures. (Figs. 1 and 2.) The presence of these bone and joint lesions, unknown to the patients, is significant and points to the relative analgesia of these conditions and also indicates the reason for the absence of bone atrophy in the roentgenograms.

To attempt to differentiate uniformly between congenital and acquired luetic arthritis is quite impossible, although each type offers helpful differential characteristics, which when considered with

Figure 4. Syphilitic arthritis complicated by tuberculosis. The presence of syphilis is indicated by osteosclerosis and periostitis.

the age, history, etc., will aid in most instances in establishing the type.

Characteristic roentgen appearances have been described most clearly and comprehensively by Baetjer and Waters[9], and nothing can be added to their complete and classic description: "In congenital lues the joint involvement is generally multiple, and there is periarticular swelling. The cartilaginous surfaces of the joint are intact, but there are marked disturbances in the epiphyseal bone region. Here the changes take place partly in the epiphyseal line and partly upon the diaphyseal side of the epiphyseal line. The process is distinctly a destructive one. There are localized areas of softening and destruction just beneath the periosteum at the point where the periosteum stops and the cartilage of the joint begins. These areas of destruction are circumscribed and punched out, suggesting the appearance of having been bitten out by a rongeur forceps. They start at the epiphyseal line and extend back into the bone proper. The epiphyseal line in the undestroyed

portion appears perfectly normal. Atrophy is generally absent, and there is practically always more or less periostitis." "Luetic arthritis shows marked periarticular swelling, thickening of the synovial membrane and fluid in the joint. This type does not go on to cartilaginous destruction, consequently when the disease subsides it leaves a perfectly normal joint. The *x*-ray appearance, as far as the joint is concerned, is identical with that of an acute polyarticular rheumatic or a traumatic joint. Fortunate-

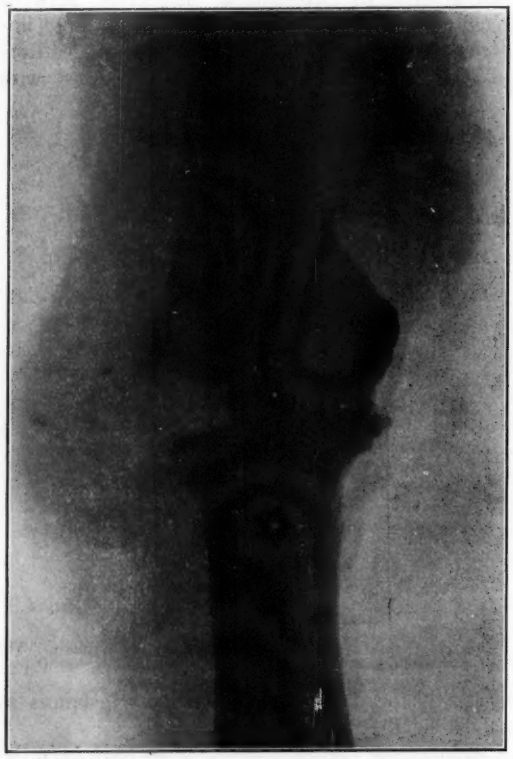

Figure 5. Destructive osteo-arthritis type of syphilitic arthritis. Marked destruction of the articular ends of the bones with some osteoperiostitis. Bone atrophy, proportional to the destruction, is obviously absent. Osteosclerosis predominates. Roentgenologically simulates Charcot joint.

ly, in the majority of cases of this type of arthritis, it is generally accompanied by bone change, and while this change is not in the joint itself, it is fairly characteristic and may be recognized as a small area of periostitis formed just at the chondroperiosteal junction. This periostitis, in conjunction with periarticular swelling and fluid in the joint is fairly characteristic of a luetic lesion. This lesion is seen most frequently in early adult life. The question will immediately be raised that periostitis occurs with other forms of acute joints. This is perfectly true, but the differential point is that in such joints there is always destruction in the joint itself, while in syphilis the periostitis is associated with no joint destruction."

Perhaps insufficient emphasis has been given the question of the absence of bone atrophy in luetic arthritis. Usually these lesions are free from incapacitating pains, consequently they are used at least moderately, so that atrophy of disuse is usually absent, or absent to a degree that is appreciably incompatible with the duration of the arthritis. This characteristic, in the presence of a chronic arthritis, with synovial and periarticular thickening, should immediately raise a suspicion of syphilitic arthritis, even if no focal periostitis can be discovered. In the detection of this pathognomonic periostitis at the chondroperiosteal junction, it is important to remember that

Figure 6. Charcot neuroarthropathy of first metatarso-phalangeal joint.

this area is usually very small and circumscribed, and that in the routine antero-posterior and lateral exposures, only four profiles are visualized. For this reason, where lues is suspected, two or more oblique exposures should be made. The routine views may be entirely negative for definite specific characteristic findings, whereas the oblique views show the focal periostitis at the chondroperiosteal junction, thus identifying the arthritis as syphilitic, (Fig. 1.) This frequent experience has influenced me to make routine oblique views, in all cases of synovitis without atrophy that have existed for more than six weeks; and this modification in technic has disclosed localized patches of periostitis and small gummata that otherwise would have been overlooked.

For clearness of description, as well as brevity, the following classification is submitted. It is based upon the most easily differentiated roentgenological and therefore pathological features:

1. Simple congenital types, with gouging out

of the ends of the diaphysis, frequently multiple and symmetrical. The necessity for brevity permits me only to refer to this type, which is so admirably described with complete differential diagnostic considerations by Baetjer and Waters.

2. Synovial types, which may be either single or multiple, congenital or acquired. They may have demonstrable focal periostitis at the chondro-periosteal junction or not, but they are of long duration without commensurate atrophy. Synovitis of more than two months duration,

Figure 7. Symptomless Charcot neuroarthropathy of lumbar spine. Accidentally discovered in course of roentgenological examination for Charcot neuroarthropathy of hip.

without atrophy, should be suspected as syphilitic unless proven otherwise.

3. Simple osteo-arthritic types. A dense synovitis is usually present. As usual, atrophy is conspicuous by its absence. The articular ends of the bones forming the joint are the seat of a syphilitic ostitis or periostitis or osteoperiostitis.

4. Destructive osteo-arthritic types. Fortunately this type is encountered infrequently (three percent. of my series). Extensive destruction of the articular surfaces take place. A dense synovitis is also present. Again the atrophy is only slight, not commensurate with the amount of destruction and the duration of the condition, which serves to make a combination that should immediately cause suspicion of syphilis.

Both the synovial and the osteo-arthritic type should have the benefit of a careful physical examination of the bones and joints of the body, with roentgen examination of any suspicious part. Often syphilis in symptomless tibias and other bones has been discovered thus. For example, a recent roentgenological examination of a child showed bilateral congenital saber tibias and definitely established the syphilitic character of a simple synovitis of the hip, of long duration, with only very slight atrophy.

5. Syphilitic arthritis with secondary infection. The greatest possibility of error in diagnosis is in this type, where the combined effects of multiple infections give decidedly atypical appear-

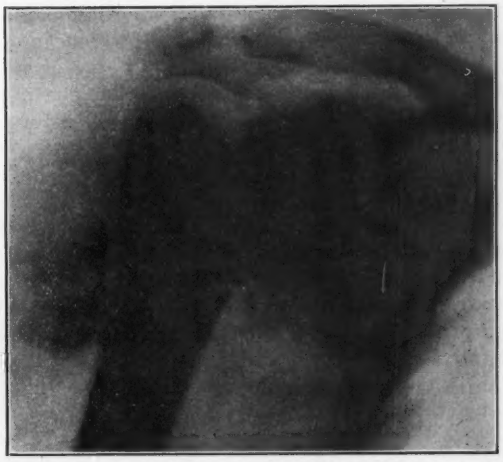

Figure 8. Neuroarthropathy of shoulder—probably due to peripheral nerve injury. Neurological and serological examinations entirely negative.

ances. In these cases atrophy is usually present, and to a marked degree, so that one of the most dependable characteristics is lost. (Fig. 4.) Synovitis with periarticular swelling is usually present. The finding that serves to identify the presence of lues is an osteo-periostitis in the ends of the diaphysis of the adjoining bones. Mention must be made of the effect on the roentgenographic appearance of a syphilitic arthritis of a severe trauma into a joint, or of immobilization over a long period. Here allowances must be made for the osteoporosis following the immobilization, with this exception, that the joint will be found to correspond in most respects to what has been described before.

The destructive type of syphilitic osteo-arthritis is differentiated sometimes with great difficulty roentgenologically from very early, Charcot disease, which it may resemble very closely in many respects. (Fig. 5.) One case has been observed recently, which shows typical roentgenological as well as physical findings of a Charcot

joint, without, however, the neurological findings of tabes. The reverse is also true—tabes may be present with joint affections of the synovial or osteoarthritic types. (Fig. 6.) In these cases, extensive destructive arthropathies may supervene very rapidly. (Fig. 7.) The presence of such extensive joint destruction and disorganization, in the absence of tabes, suggests strongly that a severe infectious or traumatic peripheral neuritis involving the trophic nerves has contributed at least partially to some of the joint changes. This brings up the question of certain phases of neurotrophic joint disease, which has been discussed recently by me, conjointly with Charles Rosenheck.[10] We called attention to a type of neurotrophic arthropathy, which is not caused by tabes, syringomyelia, spinal anomalies or injuries, and which in all probability is caused by peripheral nerve disease or injury. This communication included a case report, the second of its kind that could be found in medical literature. Since our publication, I have encountered a second case that presents a neurotrophic elbow joint, typical, both physically and roentgenographically, with entirely negative neurological and serological examinations, as well as negative history and negative therapeutic reactions. The possible existence of such neurotrophic joint disturbances with conflicting negative neurological data must be kept in mind and the condition must be recognized as a clinical and pathological entity. (Fig. 8.)

SUMMARY

1. Syphilitic arthritis manifests itself roentgenographically in definite individual characteristic ways in all stages.

2. A classification is submitted based on roentgenographic considerations.

3. Synovitis, of more than two months duration, without atrophy, should be considered syphilitic, until proven otherwise.

4. Pathognomonic foci of periostitis at the chondro-periosteal junction may be demonstrated by oblique roentgenograms.

5. Severe destructive types of syphilitic osteoarthritis simulate early Charcot joints and suggest possible early peripheral nerve disease as a factor in their production.

6. Negative Wassermann reactions are of no diagnostic value in the face of positive roentgen findings.

7. Syphilitic disease of the bones and joints may exist without symptoms, (my series includes 11 cases accidentally discovered in the course of *x*-ray examination of other affections.)

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SCURVY IN YOUNG CHILDREN: ITS DIFFERENTIAL DIAGNOSIS FROM SURGICAL AND OTHER DISEASES*

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Although, except for minor and trifling differences, the etiology and pathology of scurvy are essentially the same in adults, children and susceptible animals, nevertheless, the clinical manifestations and course of the disease in young children present special features and peculiarities. In the early days infantile scurvy, although observed clinically, was considered a form of rickets, and, unfortunately, the use of such terms as "acute rickets", "hemorrhagic rickets", "scurvy rickets," obscured the true nature of the condition. Glisson, however, as far back as the 17th century, in his "Treatise on Rickets" emphasized that rickets and scurvy were disorders separate and apart, although sometimes combined, and he clearly differentiated them from the standpoint of symptoms, anatomical findings and treatment. Today, we know that rickets and scurvy are distinct and different conditions as regards, etiology, symptoms, pathology, *x*-ray findings and therapeutic response, albeit both conditions are at times found in the same individual.

We owe our present knowledge of the pathology and symptom complex of infantile scurvy largely to the English physicians, Cheadle (1878) and Gee (1881) and particularly to Sir Richard Barlow (1883). In fact, on the continent, the condition is known as Barlow's disease or Möller-Barlow's disease.

The older observers recognized that the symptoms of scurvy occurred when certain articles were absent from or limited in the dietary and that these symp-

*Read at a meeting of the Clinical Society of the Hospital for Joint Diseases, December, 1924.

toms disappeared upon the administration of fresh fruit juices (especially citrus,) vegetables and meat. It remained, however, for the newer investigations to show that scurvy is a deficiency disease in the sense that it is due to a diminution or absence in the diet of a vital factor in the food supply, for as Hess states "unless the diet of man includes food possessing this antiscorbutic property, scurvy will develop in time."

The wave of infant hygiene propaganda which has swept over this country during the past two decades has made infantile scurvy far less common than it was some 25 years ago. At that time, while assisting and later succeeding Dr. Koplik at the Good Samaritan Dispensary, scarcely a month passed without several cases being seen. Today it is the exception—as years ago it was almost the rule—for parents to withhold orange juice and fresh vegetables and other antiscorbutic food from children in the latter half of the first and during the second year. It is this common practice of giving infants and children a more mixed and varied diet, rather than a onesided or restricted diet, that is largely responsible for the comparative rarity of this condition these days. Then too, despite its occasional observation, it is certainly rare to see nowadays the severe and aggravated types of yesteryears, and this is due in no small measure to a better understanding and appreciation and a more ready diagnosis and prompt treatment by the profession.

Of course, if we picture to ourselves infantile scurvy in the light of the older text-book descriptions as meaning a child with profound anemia, debility, bluish, swollen, hemorrhagic and bleeding gums, swollen, painful, tender extremities, pseudoparalysis, skin and mucous membrane hemorrhages, then infantile scurvy may be considered a rarity. If, on the other hand, we appreciate that restlessness, irritability, pain or discomfort on handling, with or without slight tenderness over the extremities, with perhaps a few erythrocytes in the urine, mean the initial stage of scurvy, then this disorder is not so uncommon. If, furthermore, we realize that lack of desire for food, stationary or loss of weight, pallor, restlessness, edema of the eyelids, rapid pulse and respiration, perhaps slight pain on manipulation in an infant fed on the proprietary foods, condensed milk or heated milk, may be the forerunner of scurvy or, better, is scurvy in the prodromal stage, then this disorder is more frequent than generally recognized. And, finally, if we keep constantly before us that every infant who is given a onesided feeding, lacking in antiscorbutic substances, is a potential case of scurvy, then we have at our command the most important aspect of the whole scurvy problem, namely prophylaxis.

In the light of the newer etiology and pathology, infantile scurvy may be defined as a metabolic, nutritional disorder, caused by a prolonged deficiency or absence from the dietary of one of the essential food factors—water-soluble vitamin C, and characterized by anemia, restlessness, debility, edema, changes in the bones and a hemorrhagic diathesis leading to hemorrhages in various parts of the body with predilection for the gums, subperiosteum and bone marrow, and not infrequently in the skin, mucous and serous membranes and internal organs.

Once scurvy has manifested itself, unless specific dietary treatment is instituted, it progresses steadily, its symptoms and signs become aggravated and the patient succumbs as the result of exhaustion, cardiac failure or intercurrent respiratory or gastro-intestinal disease. The disorder is never acute in its onset, although it may appear so from the symptoms when the patient is first seen. Premonitory or insidious symptoms always precede the obvious signs of an apparently acute onset and unless these are checked by appropriate diet, unfortunate symptoms of varying grades of severity follow sequentially.

Accordingly, therefore, as the patient is seen at one period or the other, we note the following grades or stages of scurvy:

1. Premonitory, prodromal or prescorbutic stage.
2. Incipient or subacute scurvy.
3. Acute scurvy—mild, moderate or severe.
4. Fatal scurvy.

Let us follow a case through these various stages. Given an infant of six to ten months of age fed for a long time on restricted diet, let us say one of the proprietary foods, condensed milk or high diluted pasteurized or sterilized milk, bread-stuffs and cereals (without supplemental fruit juices, green vegetables, beef juice or potato), whose weight remains stationary or who is losing weight for several weeks, who becomes pale, fretful, restless, whose food disagrees with him, whose pulse and respiration are increased and who shows a slight puffiness under the eye-lids, and you have, as a rule, an infant in the prescorbutic or latent stage, proof of which is afforded by a complete change for the better upon the administration of orange juice. Fail to interpret these findings and there follow irritability, peevishness, pain and tenderness on manipulation, disinclination to move the extremities, discoloration of the gums, and red blood cells in the urine microscopically or a few petechial spots on the neck and back—a case of incipient or subacute scurvy. Overlook or minimize these findngs and there are superadded exquisite tenderness of the limbs, swellings in the neighborhood of joints, pseudoparalysis, hemorrhagic, swollen gums, petechiae or ecchymoses, edema, perhaps hematuria—a well developed case of scurvy. If, after all this, proper treatment is not instituted, there follow debility, asthenia, sub-

periostal hemorrhages, bleeding from and into internal organs, skin and mucous membranes, immobility of extremities, bronchopneumonia or gastroenteritis and death—the fatal form of scurvy.

These symptoms will follow just as sure as does the day the night, unless an antiscorbutic regimen is given the infant. And just so sure will they be prevented and just so sure will the symptoms and signs be halted at one or the other stage—unless indeed the condition is too far advanced—if only a sufficient and liberal supply of orange juice or other antiscorbutic food is administered.

It has been stated that the onset of scurvy is never acute. The reason for this is clear when it is remembered that it takes several months—according to Hess, about six months—of food deprivation to establish a negative vitamin balance before the individual manifests signs and symptoms of scurvy.

Whether the lack of this essential food factor, vitamin C, is in itself the cause of scurvy or whether its absence "leads to the development of substances which in themselves occasion the symptoms of the disease" or whether this lack leads to diminished resistance with its attendant dangers of infection has not been definitely established. This much is certain, failure to supply the organism with sufficient vitamin C is directly or indirectly responsible for the train of scorbutic symptoms.

Apart from the relation of diet to scurvy we must recognize, as pointed out by Heubner and Flesch, that there exists a predisposition to Barlow's disease as shown by the fact that among a group of children living under the same environmental and dietetic conditions, some few will get scurvy while the majority will escape. Furthermore it has been observed that in cases of twins, fed and living exactly the same, one will escape and the other will become scorbutic. This disposition to scurvy is classified by the Germans under the heading of constitutional *Minderwertigkeit*. This explains why scurvy is more prevalent in some families than in others, just as it explains why in certain families the respiratory or intestinal tract, the nervous system or the skin is the vulnerable point of attack.

Hess calls attention to the fact that the exudative diathesis type of child is more readily affected with scurvy, more predisposed in other words, than the normal child, and concludes by saying "the exudative diathesis implies not only a diathesis for exudations but also for the development of scurvy."

The relation of scurvy to age and social status has import from the clinical standpoint. Scurvy in children is most commonly observed in the last half of the first year, over three-fourths of the cases being observed between the 6th and 10th month. It may occur in the later ages of childhood, but each succeeding month of life with the greater probability of increased variety of food, makes the likelihood less. It has been reported as early as the third or fourth month; the earliest age at which it has been seen by Hess was four and one-half months. Holt says "I have never seen and have never known of a case of scurvy in a child over two years old who was living under normal conditions."

It may seem strange that infantile scurvy is, if anything, more common among the well-to-do and the middle class than among the poor. In fact, Still says "It is seen in private practice even in the best surroundings quite as often as among the poor." But among the poor there has always been more breast feeding and their children have not been fed "ultra-scientifically" as Holt was wont to say, but have been given early and sometimes often a variety of table articles or a "taste of everything," which supplied the necessary antiscorbutic content to the diet.

Scurvy, more than rickets, is essentially a food disorder, hence the better environment of the rich offers no protection. Being a food disorder it is natural to inquire under what conditions of feeding and with what kinds of food scurvy is likely to develop. The answer is simple—with all kinds of foods, under certain conditions and in certian infants. Thus infantile scurvy has been observed in infants fed upon breast milk, raw, sterilized, pasteurized, peptonized cow's milk, condensed milk, proprietary foods and others. It is true that by far the greater number of cases have followed prolonged exclusive feeding with proprietary foods, heated or re-heated milk and condensed milk but not all by any means. It is conversely true that the probability of scurvy developing in a nursing baby or one fed on a proper quantity of raw milk is far less than in one fed exclusively on pasteurized, condensed or sterilized milk, or proprietary foods, since as a rule the amount of antiscorbutic vitamine is greatest in the both groups of the fore-mentioned in the order named.

There are many foods rich in antiscorbutic substances which should be given infants. Food, however, is so important a part of this subject that I prefer to leave it for fuller discussion when I take up the questions of prevention and treatment.

It would seem as if scurvy in children, at least in active cases should be easily diagnosed, and yet errors are altogether too common. The usual thing is that scurvy is diagnosed as some other condition rather than the reverse. The greatest cause of error, apart from failure to constantly keep the condition in mind when dealing with infants, is the unfortunate diagnostic emphasis given in text-books to the condition of the gums and tenderness and swelling of the limbs as necessary accompanying signs and symptoms of the condition, as well as failure to elicit

a full history of the feeding and to make a complete physical examination. The latter point is emphasized by Pfaundler and Schlossman in the statement "the entire child should be examined." Furthermore, in obvious and advanced cases too much attention is focused upon local objective findings as evidence of a lesion in that particular part or organ without considering associated and collateral symptoms. The latter statement will become more evident when differential diagnosis is considered.

The type of diagnostic error varies naturally with the stage of the disease and errors in this disease are serious,—1. because of unnecessary and often harmful operative interferences which have been undertaken under misapprehension; 2. because delayed treatment may result in a fatality; 3. because a rapid response with alleviation and cure promptly follows proper dietetic treatment.

The conditions for which infantile scurvy have been mistaken are strange, varied and numerous. They comprise a group showing little if any, correlation and yet the errors are explainable when the pathology of the condition is considered.

Before considering differential diagnosis, I should like again to emphasize four important and basic leads in diagnosis:

1. Full and complete history of feeding
2. Thorough physical examination
3. Roentgenological examination of the extremities of both sides
4. Therapeutic test—response to antiscorbutic feeding.

Rheumatism: Rheumatism is a disease of later childhood and is rarely observed under two years of age. Six to fifteen months is the age of selection for scurvy and the age of exception for rheumatism. A case of infantile scurvy so far advanced as to cause suspicion of rheumatism would have discolored or bleeding gums, superiosteal hemorrhages, extreme tenderness of the extremities and, likely, red blood cells in the urine. The existance of the "Trümmer Zone" at the epi-diaphyseal junction of the long bones and the disappearance of symptoms with proper antiscorbutic diet, without drugs, dispel all doubt.

Trauma: Inability to move the limbs and the presence of "black and blue" spots on the limbs or body bring the infant to the physician with the parent's diagnosis or thought of injury. Scurvy patients, by reason of the fragility of the bloodvessels, are more susceptible to ecchymoses following slight injuries than normal children. Concomitant symptoms—pain or discomfort on handling, fretfulness, discoloration of the gums, the *x*-ray and the therapeutic test make differentiation easy.

Infantile Paralysis: It is natural for parents who find that the infant is disinclined to move its limbs to think first of paralysis of some sort and especially infantile paralysis. This is a common error on their part and not an uncommon one on the part of physicians. Infantile paralysis is, however, a febrile disease, acute in its onset, not exquisitely painful or tender except in the hyperesthetic form. There is absence of the hemorrhagic tendency, of swellings along the long bones, of edema, of pain on deep pressure. There is disinclination to use the extremities in both conditions, but in scurvy it is because of pain, separation of the epiphysis or the weight due to muscle infiltration or subperiosteal hemorrhage, while in poliomyelitis it is due to muscle paresis. Here again, the roentgen findings and the therapeutic test come to our aid.

Osteomyelitis: Occasionally, especially where there is marked swelling and edema at the ends of the long bones due to subperiosteal hemorrhage with elevation of the periosteum, a differentiation from osteomyelitis must be made. Remembering that scurvy is a hemorrhagic disease, that the involvement is usually bilateral and symmetrical, although this is not absolute as Barlow states, that the temperature is usually low or absent unless complications exist, and remembering that osteomyelitis is usually associated with high temperature and with high leucocyte count as a rule, and is usually unilateral, differentiation should not be difficult. If the question of operation arises, it is prudent to wait upon the *x*-ray findings and the administration of orange juice. Two or three days use of the latter will decide whether operative interference is indicated.

Sarcoma: Why sarcoma or a malignant osseous growth should be suspected in cases of scurvy may be hard to understand except for those who have seen large subperiosteal partially organized scorbutic hemorrhages over one extremity, more frequently the lower end of the femur. Close and careful examination usually discloses involvement of the opposite side, pain, tenderness, slight swelling, thickening or resistance, as well as evidences of hemorrhage elsewhere—gums, urine, palate and skin. It is the apparent unilateral involvement that has caused most of the errors. But if the thought of sarcoma occurs, why hurry? Our friend, therapeutic test, is always with us and will accomplish great good when the knife may cause irreparable harm. When in doubt remember that an orange a day may keep the surgeon away. Holt mentions two cases of scurvy operated upon by eminent surgeons—one for sarcoma, the other for ostitis.

Syphilitic Epiphysitis: There are several points of similarity between this condition and infantile scurvy: both affect infants; both are painful; both are associated with pseudoparalysis. But there the similarity ends. Scurvy is very rare before the

sixth or seventh month, syphilitic epiphysitis is rare after the third month. Scurvy is usually bilateral, Parrot's paralysis usually unilateral. The pseudoparalysis of scurvy is more common in the lower extremity, that of syphilis in the upper. Scurvy is a hemorrhagic, nutritional, deficiency disorder, syphilitic epiphysitis an infectious, constitutional disease. Other signs of syphilis are usually present and the *x*-ray findings are different and characteristic for each. The therapeutic test removes all doubt.

Hemorrhagic Nephritis: There are cases of scurvy in which the first sign appears to be hematuria. Sometimes the urine is dark, sometimes the blood is microscopical. With this may be associated oliguria, not uncommon by the way, in the common variety of scurvy. With this hematuria one may find casts and pus in the urine. A history of the feeding, careful search for other signs, condition of the gums, *x*-ray findings of the bones, makes the differentiation. These cases respond to the therapeutic test—the use of orange juice, etc. Upon the administration of orange juice, oliguria in scurvy gives way to normal secretion of urine not, in my opinion, because orange juice is a diuretic, but because it controls the transudation of serum through the vessel walls. The microscopical appearance of red blood cells in the urine is due to diapedesis or to petechiae in the kidney pelvis.

Intussusception: It seems a far cry from infantile scurvy to intestinal obstruction and yet, no less authorities than Still, Holt, and Erdman have encountered instances where the question arose. Still mentions the case of a child with blood and mucous in the stools and vomiting, with flaccid and hollow abdomen and "in the direction of the transverse colon it showed an elongated sausage like tumor which on palpation was firm and seemed to vary in hardness at intervals during palpation." Under appropriate antiscorbutic treatment the child improved and the abdominal tumor disappeared.

The importance of quick differentiation under such conditions requires no emphasis, for intervention in intussusception to be successful must be prompt—the earlier the better.

Here, I might mention also cases of scurvy with muco-hemorrhagic stools in which a diagnosis of colitis is not infrequently made, unless other signs of scurvy are sought. The discharges are due in part to diminished tolerance and resistance and in part to edema of the intestinal mucosa and permeability of the bloodvessels. Orange juice, despite its supposed laxative effect, cures the condition.

Rickets: Scurvy and rickets are separate entities —as regards etiology, pathology, symptoms, *x*-ray findings and therapeutic response. "The scurvy tendency to hemorrhage and the prompt curability by fruit juices have no counterpart in rickets." (Holt) unless we accept a similarity between the prevention of rickets by the use of cod liver oil and that of scurvy by orange juice. Both, however, are essentially deficiency nutritional diseases, in both the age of susceptibility is 6-15 months, but in rickets environmental factors play a rôle in causation.

The occurrence of both conditions in the same individual is due to the fact that a feeding program which omits one vital food factor usually omits other vital factors as well. It is not an uncommon experience to find that the most aggravated cases of scurvy are free from any evidence of rickets and it is interesting to record that Still says, "potato, which is a soverign remedy for scurvy, will produce rickets if it forms a large part of the diet for a long time."

In rickets there may be tenderness over the long bones on handling and even pseudoparalysis, while in scurvy rib rosary was noted by Barlow. Barlow called attention to "weakness of the back" in scurvy and furthermore states that in some cases "the sternum with the adjacent costal cartilages and a small portion of the contiguous rib seems to have sunk bodily back *en bloc*, as though it had been subjected to some violence which had fractured several ribs in front and driven them back." The *x*-ray findings and the therapeutic test determine the diagnosis.

Nasal Diphtheria: A persistent muco- or sero-hemorrhagic discharge from the nose in children always excites a suspicion of nasal diphtheria. In the absence of membrane and with a negative bacteriological finding it is well to think of infantile scurvy because, as Hess and recently Nassau and Singer have pointed out, hemorrhagic rhinitis is often the forerunner of scurvy. On the other hand, scurvy renders the nasal mucous membrane susceptible to the diphtheria bacillus so that nasal diphtheria sometimes is a forerunner of scurvy.

Spondylitis: Occasionally in scurvy, the spine is exquisitely tender, resulting in rigidity and fixation. Such cases when presented first to the orthopedist, as they usually are, may excite a suspicion of spinal disease. Taylor, Kerley and LaFetra have reported such cases. The differentiation is made along lines noted in the aforementioned captions.

The x-ray findings of infantile scurvy are typical and present two characteristics:

1. A shadow at the epi-diaphyseal junction known as the white line of Frankel or "Trümmer zone" seen in mild and advanced cases;

2. Spindle form shadows of various sizes when subperiosteal hemorrhages are present, especially when calcium salts have formed beneath the periosteum. Occasionally, without subperiosteal hemorrhage, shadows due to diapedesis or hemorrhage into the muscles are observed.

The Trümmer zone is the result of dead bone cells

following hemorrhage and it consists of an irregular, distorted shadow usually wider in the center than at the ends. It is essentially a widened preparatory calcification zone. Differentiation must be made from luetic epiphysitis and rickets. In lues the epiphyseal shadow is wider and less distorted and irregular and there is usually the parallel ossifying periostitis or osteoperiotitis along the diaphysis.

In rickets the epiphyseal line is less defined, is irregularly fringed and often shows a cup-like depression. In rickets the *x*-ray findings are found simultaneously in the lower epiphyses of all the long bones, whereas in scurvy the epiphyseal shadow is usually limited to the lower or upper extremities. While the epiphyseal shadow in scurvy is constant, in mild and severe cases, the *x*-ray will also at times detect intramuscular edema and hemorrhage, epiphyseal separation, pericardial effusion and enlargement of the right heart (Erdheim, Hess). Erdheim considers hypertrophy of the right ventricle, typical of scurvy. Hess says of the "white line"; "Its diagnostic value has been greatly exaggerated. We have found it frequently missing when the disorder is advanced and it persists for months after a cure has been effected."

The pathology of scurvy centers about a hemorrhagic diathesis which results in bleeding to an extent and degree depending upon the stage of the disease, its severity and the susceptibility of the patient. Thus hemorrhages of a petechial or ecchymotic nature may appear upon the skin or scalp, the gums, hard palate, eyelids, orbits, or in the kidneys, intestines, pleura, pericardium, periosteum and bone marrow. Why these hemorrhages in scurvy? In the so-called hemorrhagic diseases, that is, various forms of purpura, hemophilia and scurvy, we look to the pathology in the blood itself, the blood-forming organs or the bloodvessels. Recent hematological investigations have thrown much light upon the differentiation of these diseases as viewed from the standpoints of coagulation time, bleeding time, platelet count and capillary resistance or Rumpel-Leede test, in other words, from the standpoint of the physical characteristics of the blood and vessels rather than from the morphological characteristics or changes in the blood cells. The present day opinion as regards the etiology of the hemorrhagic tendency in scurvy is best summed up by Hess as follows: "hemorrhages are attributable to a disturbance of the vessel walls rather than to a defect of the factors concerned in the normal coagulation of the blood." There exists then, in scurvy, a fragility of the vessel walls and an increased permeability, the former permitting of hemorrhages because of rupture of capillaries and venules, the latter of diapedesis of serum and erythrocytes. It is the latter which explains the edema into the muscles and tissues.

This fragility of the vessel walls has been called angiomalacie by Aron and angiodystrophy by Meyer and Nassau. The latter consider petechiae the result of this vessel dystrophy and, situated on the face, an early symptom of infantile scurvy. Hess states "we would, however, add to the category of early signs of this disease, the development of numerous petechial spots." The following table is an interesting, schematic presentation of the important factors concerned with bleeding in hemorrhagic conditions:

Hemorrhagic Diseases—(Ottenberg and Hess)

	Coagulation Time Prolonged (Normal 5-10 mts.)	Prolonged Bleeding Time (Normal 2-5 mts.)	Blood Platelets Diminished (Normal, 200,000).	Tourniquet Test Present Capillary Resistance.	Skin Puncture.
Hemophilia	+	+*	—	—	—
Purpura Simplex	—	—	—	+	+*
Purpura Arthritic	—	—	—	+	+*
Purpura Visceral	—	—	—	+	+*
Purp. Hemorrhagica	—	+	+	+	+*
New-Born	+	+*			
Scurvy	—	—	—	+	+*

*Variable.

The bone changes consist of hemorrhages within the marrow and beneath the periosteum. The normal lymphoid marrow undergoes degeneration and becomes poor in cells, less vascular and more homogeneous, osteobastic activity is diminished and new bone formation diminishes or ceases. As normal bone absorption continues there results an abnormal thinness and less density of the shaft especially in the region of the epiphyses. The latter leads, even with slight trauma, to fracture or epiphyseal separation.

It is a significant fact that hemorrhage into joints —hemarthrosis—is the great exception in this condition. The swellings and ecchymoses are almost always about, not in the joints.

Prevention and Treatment: Infantile scurvy is a readily preventable disease. There is nothing mystical about its etiology. It is a nutritional metabolic disorder due to failure to supply a particular organism with a sufficient amount of one of the health- and life-determining food factors—water soluble vitamin C. It is, in other words, an avitaminosis or a deficiency disease. Mention has been made above of "sufficient amount" and here it must be emphasized that this is a relative matter. Just as different infants of the same age require varying amounts of proteids, fats and sugar, depending upon their tolerance, so different infants require varying amounts of antiscorbutic vitamin to prevent or even cure scurvy. Fortunately, we know that all vitamins are so potent that only small amounts are necessary to protect or

produce effects. I know of no method of quantitative determination of this vitamin, but no one should lull himself into a sense of security by feeling that because an infant receives some antiscorbutic food, he is necessarily protected against scurvy. It is only by careful watching of all infants, irrespective of feeding, for premonitory or preparatory signs and symptoms that scurvy can be prevented. Hess expresses himself as follows on this question: "Every individual requires a certain amount of antiscorbutic substance in his dietary. How much of this type of food is required, in other words, what is the antiscorbutic minimum for the individual is just as little known as is the minimum requirement of other so-called vitamins."

Holt says "Evidently the body has a sufficient store of this vitamin C to meet its needs for a considerable time, for symptoms of scurvy do not usually appear for some months after the deficiency diet has been taken." Since the prevention and treatment of infantile scurvy are entirely dietetic it would be well to discuss, briefly, the question of feeding in these relations. Our premise is that scurvy in infants is due to a lack or deficiency in the food of the antiscorbutic vitamin water soluble C. This vitamin, like its sisters, fat soluble A and water soluble B, are not manufactured in the body, but are ingested with the food intake. Since vitamin C is vital for the prevention and treatment of scurvy it behooves us to learn those articles of food which are rich in its content and what methods of industrial or domestic preparation, preservation and storage destroy or vitiate it.

Probably, no one has analyzed and investigated this question more extensively or minutely than Alfred Hess. From him we learn that fresh foods are richer in vitamin C than "dead" ones, raw milk richer than aged, sterilized, peptonized, condensed or pasterized milk, or proprietary foods, fresh vegetables richer by far than dried or preserved ones. orange and lemon juice richer than banana or prunes. He has also taught us that fresh or strained, canned tomatoes as well as potato are potent antiscorbutic agents.

It cannot be gainsaid that scurvy has occurred in infants at the breast and in those fed on raw milk, neither of which have been subjected to any artificial process likely to lower or destroy vitamin C. The reason for this is clear when it is remembered that the vitamin content of these milks depends upon the vitaminic intake of the mother or the cow, and this lacking in the diet of one or the other will result in a milk deficient in vitamin C.

The fact is that scurvy has occurred in infants fed on a variety of food, but usually when the diet was restricted or one-sided. For this reason it becomes the duty and responsibility of those in charge of infants to supervise carefully the dietary so that it is well balanced and contains not only a proper proportion of the organic nutrients—proteids, carbohydrates, fats and salts, but also of all classes of vitamins. For vitamins, as C. H. Lavender states "are the ignition spark, the energy of which is insignificant, but without which the engine cannot run."

The sovereign remedy in scurvy is orange juice. To prevent and cure I am for orange juice. After that I am for more orange juice. I hold a dietetic creed, that all infants, no matter how fed—breast or otherwise—shall receive orange juice once daily from the second or third month of life in doses beginning with twenty or thirty drops and increasing until the juice of a whole orange is taken daily by the sixth or seventh month. Orange juice, like the tomato, does not lose its antiscorbutic potency by boiling. This is a simple, safe and certain preventive. I might mention here that this early administration of orange juice is just as routine a practice with me for the prevention of scurvy as is the administration of cod liver oil in doses of twenty drops to one drachm three times daily to all infants from 6-12 months during the months of October to March, for the prevention of rickets.

With the diagnosis of scurvy established or suspected, the administration of antiscorbutic food is indicated. The type of food varies, of course, with the age of the patient. Orange juice, raw milk, baked, mashed or creamed potato, fresh boiled or strained canned tomatoes, beef juice, fresh spinach, fresh young carrots, lettuce, yellow turnip, afford a variety. The amount of vitamin C in raw milk is small. Chick and Hume found that a guinea-pig fed on vitamin C-free food required 100 c.c. of milk daily to protect it against scurvy, while 1½ c.c. of orange juice gave complete protection. For quick action orange juice is the diet of choice.

The early recognition and early treatment of scurvy are important because this is not a disorder that runs a limited course, but rather one that, if unrecognized and untreated, becomes progressive and sometimes fatal.

Infantile scurvy, like diabetes, is one of the easiest diseases to diagnose—if one ony thinks of it; errors in diagnosis are most frequently due to failure to keep the condition in mind. Brought face to face with a child of from six to fifteen months who shows evidence of pain or tenderness on handling and inability or weakness of or disinclination to move the limbs, the first thought should be scurvy and the next orange juice.

There are few more satisfactory responses in the realm of medicine than the picture of an ailing, suffering, restless, unhappy, motionless child brought back to rest, quiet, sleep, comfort and happiness in two or three days by the simple use of orange juice

or other antiscorbutic food. The action is so dramatic and so prompt as to leave no doubt in one's mind of cause and effect. "The specificity of action of these substances (vitamins) is quite as great as that of antitoxin in diphtheria or thyroid extract in cretinism" (Holt.)

The treatment of infantile scurvy does not terminate with the relief or cure of the symptoms. A symptomatic cure is not a pathological or nutritional cure. The hemorrhagic effusions do not subside so quickly and the body cells and vessel walls do not return to normal at once, as shown by the occasional recurrence of hemorrhage or symptoms later and the presence of the capillary resistance reaction often for weeks after the relief of symptoms.

A close supervision of the diet, rest, fresh air and sunshine and iron in the form of syrup of the iodide of iron or saccharated carbonate, are indicated.

Strange as it may seem a relatively large number of cases of infantile scurvy, large for these days, has been observed at The Hospital for Joint Diseases. During the past two years my associate, Dr. Pollock, and myself have seen nine cases with evident and obvious signs and symptoms and an equal number of suspected ones yielding to an antiscorbutic therapeutic test. Many cases of malnutrition presenting symptoms of scurvy in the premonitory stage are overlooked, and if these were included in a statistical enumeration, the number would be larger than that reported. After all, it may not appear so strange that we see these cases at the hospital when one considers that those observed by us were diagnosed either as rheumatism, infantile paralysis, injury or joint inflammation, and when one remembers that with the broadening conception of the proper realm of orthopedics by the laity, most deformities or limb incapacities are now finding their way to orthopedic institutions.

A case of moderate or advanced scurvy calls for the minimum amount of handling, even as regards examination by the physician. Nature asserts itself and the limbs assume the position of greatest comfort and relaxation, the lower ones in slight flexion at the hip and knee, with slight outward rotation. If pain is very severe or if there is a fracture or epiphyseal separation, splints are necessary. "Handle with care" or better "Hands off" should be the watchwords in the care of these infants.

Prevention is the keynote of modern medicine and in the prevention of scurvy as, indeed, in its treatment, three things are necessary: first, orange juice; second, orange juice; third, more orange juice and of the three, the last is the most important.

If a frightened or refractory child will not open its mouth, pass a probe between two teeth and back to the palate. Instantly the mouth will open and a gag may be slipped in.—W. M. B.

A SYSTEM OF ADHESIVE STRAPPING FOR STATIC DISORDERS OF THE FOOT

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The relief of pain consequent upon certain static deformities of the foot, usually grouped under the generic term "flat-foot", often presents a problem of considerable difficulty to the orthopedist. In this group I would place: (1) simple flat foot; (2) spastic flat foot; (3) acute foot strain. All three of these conditions arise from a disorganization of the mechanical and architectural constituents that render the foot an efficient support to, and a means of propulsion of, the body weight. In spastic flat foot there is often an added factor of infectious arthritis.

Without discussing the anatomy, etiology, or pathology peculiar to the different varieties of foot strain, this communication merely aims to present very briefly a simple method of treatment that has proved very useful in my hands in relieving the painful symptoms arising from this group of foot troubles.

The relief of pain is the primary prerequisite in the treatment of these cases. After this end has been attained, we can proceed to restore the mechanical adequacy of the foot.

By applying a strapping of zinc oxide adhesive plaster according to a method that has been devised in the orthopedic clinic of the Hospital for Joint Diseases, and the Lenox Hill Hospital, I have been able in a vast majority of the cases to relieve the painful manifestations, and to a large extent restore the normal contour of the foot, so that the relief could be made permanent by a further course of corrective exercises, or corrective flat foot braces of the Whitman type. This system of strapping aims to give support to both the internal longitudinal arch and the anterior metatarsal arch, and at the same time to throw the body weight upon the external border of the foot. It aims to restore the normal contour of the arches insofar as the pliability of the particular foot will permit. It aims to transfer much of the strain of the body weight from the overstretched and fatigued musculo-ligamentous structures of the foot, to the skin of the leg.

Any surgeon conversant with the traction method of treating fractures with Buck's extension straps, will appreciate the fact that the skin of the leg can bear a very considerable and long-continued traction strain through the intermediation of good, non-irritating adhesive plaster. Many brands of adhesive plaster exert a decidedly irritating influence on the skin. If the adhesive plaster is of a good quality, if the skin is healthy and free from lesions, and if the leg is shaved, the skin will be able to bear a very considerable portion of the strain that falls particu-

larly upon the internal longitudinal arch of the foot. The plaster must be smoothly applied and uniformly adherent, otherwise the skin may not stand the burden placed upon it.

In applying this plaster strapping we attempt to support the internal longitudinal arch by a plaster sling, which also is designed to pull the calcaneus and the mid-tarsus into inversion. At the same time, by a second series of straps, the fore-foot anterior to the Lisfranc line is carried into a moderately everted position and the anterior metatarsal arch is also reconstituted and supported.

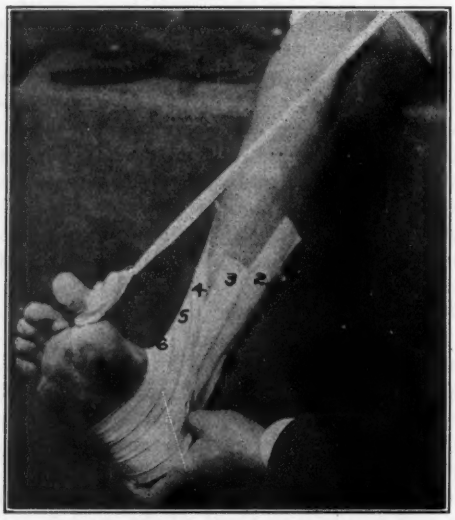

Fig. 1 The first stage in the application of the plaster, as seen from the internal aspect of the foot. The numerals indicate the order in which the straps are applied. The first two straps are applied as a unit, and control the position of the calcaneus. The inverted position of the foot is maintained by the gauze traction cord, which is held by the patient.

For the usual adult foot, eight straps of adhesive plaster are necessary. These straps are 2½ cm. wide, and long enough to extend from the external malleolus around the plantar surface of the foot to within about 10 cm. of the knee joint on the internal aspect of the leg. The length usually required is approximately 45 cm. In addition to the straps, a small piece of felt cut in a semi-oval shape is required as an anterior metatarsal pad to fit in the hollow behind the metatarsal heads. This pad is shown in Fig. 3.

The patient is seated in a chair facing the surgeon, who is also seated. A traction cord of gauze bandage is tied around the patient's great toe and given to him to hold. The foot is now placed in a position of moderate inversion, with the foot at right angles to the leg, the calcaneus resting upon the surgeon's knees. By traction on the gauze cord, the patient maintains this position. The knee is preferably held in flexion during the application of the straps.

The first two straps are applied together as a unit. They are first overlapped by half their width, and smoothly placed together along their entire length. This is done in order to give some added strength to these straps which control the position of the calcaneus. By thus doubling the straps over half of their width, the combined strap is increased in strength. This strap is applied from below the external malleolus around the plantar surface of the foot; and, exerting moderately strong traction medially, the calcaneus is pulled into inversion, and then exerting traction directly upward, the plaster

Fig 2. Same as Fig. 1., viewed from the external aspect of the foot. Note the manner in which the straps diverge, fan-like, thus distributing the traction over a wide area of skin. Note also that the entire foot is pulled into inversion by these straps.

is firmly applied to the inner aspect of the leg to within about 10 cm. of the knee. The next (third) strap should extend from the external aspect of the foot immediately anterior to the preceding strap, and overlapping the former by half its width. It is applied with moderate traction, around the sole of the foot, under the internal longitudinal arch, and to the inner aspect of the leg, diverging somewhat from the two preceding straps. The fourth, fifth, and sixth straps are similarly applied, each beginning on the external border of the foot, anterior to the preceding strap, and overlapping it by half of its width, continuing around the sole of foot under the longitudinal arch, and up the leg, the successive straps diverging somewhat on the leg anteriorly in a fan fashion. It is essential that these straps should be smoothly and uniformly applied, and some practice will be necessary to acquire the technic of applying them smoothly and with uniform tension.

After the sixth strap is applied (this strap should cross the body of the navicular bone on the internal

aspect of the foot) the patient is instructed to release the traction cord which he has been holding. The entire foot is now held in a markedly inverted position by the straps already applied. The patient, or an assistant now grips the leg above the malleoli in order to hold the strips firmly to the leg in the subsequent maneuver. The surgeon then grasps the fore-foot and forcibly everts that portion of it that lies anterior to the Lisfranc line, until the plane of the metatarsal heads is brought parallel with the ground. The felt pad described above is now placed in the depression that marks the metatarsal arch

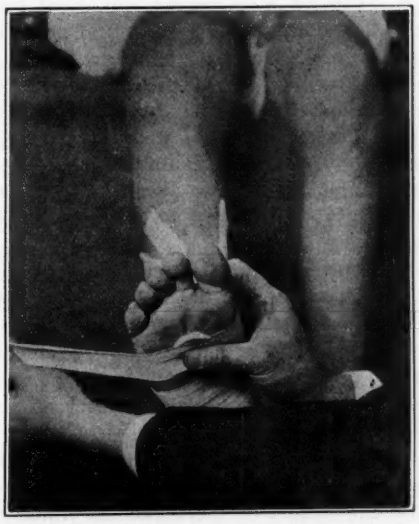

Fig. 3. The reconstruction and support of the anterior metatarsal arch, showing the application of the pad. The fore-foot is twisted from the inverted position shown in Fig. 2, to a position in which the plane of the metatarsal heads is horizontal. The fingers are depressing the head of the first metatarsal, while the thumb is exerting an upward pressure on the heads of the middle metatarsals.

directly behind the heads of the bones, and the last two straps are applied in such a manner as to retain the everted position of the fore-foot. These two straps are applied, beginning at the inner side of the foot at the middle of the first metatarsal bone, passing around the sole of the foot over the metatarsal pad, around the outer side a few centimeters proximal to the fifth metatarso-phalangeal joint, and then diagonally across the dorsum of the foot to a point below the internal malleolus. While these two straps are being applied, the contour of the anterior metatarsal arch is reestablished by pressing upward with the thumb on the felt pad. Both of these two anterior straps should pass over the felt pad, as the friction of these two straps in direct contact with the skin is sometimes very annoying to the patient. It is also essential to avoid passing these last straps over either the first or the fifth metatarso-phalangeal joint.

When the straps have been applied in the manner described, a gauze bandage should be firmly applied over the leg and foot in order to maintain a close contact between the straps and the skin. This insures uniform adhesion. This bandage may be removed after twenty-four hours.

The patient treated by the use of this strapping will complain of the traction upon the skin of the leg. In my experience, this discomfort does not last more than four or five hours. Very occasionally, however, the straps will have to be slightly loosened on this account.

It would be well if the patients could be confined to bed for a period of a week or more after the

Fig. 4. The finished strapping. Note how the anterior metatarsal straps are anchored by bringing them across the dorsum of the foot in a diagonal direction, to a point below the internal malleolus.

application of this dressing. Usually, however, he will not submit to such a regime and immediately resumes his customary routine. In these ambulant cases, I allow the strapping to remain for one week. At the end of this time it is removed, the skin is cleansed with alcohol, and fresh strapping is applied. Although the painful symptoms are often relieved after the first week, I think it best to repeat the strapping four or five times at weekly intervals. During this period, the patient should be taught inversion exercises. At the end of this period, the case can be handled in two ways: either a system of supination exercises with a view to so strengthening the muscles as to retain the corrected position of the foot, or some form of arch support. I prefer to give the patient a Whitman flat-foot brace. Later, by supination exercises, and exercises on the Zander machines, the muscles are strengthened sufficiently to permit ultimately discarding the plates. In some cases of acute foot strain, the strapping over a short period of time, affords quite permanent relief, and no further treatment is necessary.

THE MENACE AND THE MANAGEMENT OF THE CHRONICALLY DISCHARGING EAR

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A chronically discharging ear is a menace, not a mere nuisance. It is often the cause of serious, even fatal disease. Many cases of cerebral, cerebellar or subdural abscess, meningitis, perisinous abscess and sinus thrombosis, are to be seen as complications of chronic otitis in the clinics of Vienna. In this country, due to better care of the ear during the acute stage, as well as the better nutrition and therefore greater resistance of patients, we happily do not see so many of the serious complications. Nevertheless cases of markedly impaired or destroyed hearing due to chronic otitis are very common; and a fatality ascribed to idiopathic meningitis, but in reality directly traceable to the chronic otitis, is not at all rare. Proper classification and care of chronically running ears, before the onset of such complications, would undoubtedly result in the saving of many lives. Amongst many others, I may cite the following cases, which recently came to my notice, to illustrate the fatal result of neglectful treatment of chronic otitis.

An 18-year old girl was sent to the medical division of the Wiedner Spital, Vienna, with the history that 4 days before admission she was taken sick with a chill, followed by a temperature of 104°F. When signs of meningitis developed she was sent to the hospital, where she died in two hours. Autopsy showed a sinus thrombosis and basal meningitis extending from the right suppurative middle ear into the cranial cavity.

A 45-year old man with a chronic otitis developed headaches and some slight irregular fever. On account of some concomitant eye condition he was sent by his family physician to the eye department of the hospital. The ophthalmologist, not finding enough to account for the headaches, and noticing the discharging ear, notified the ear department. On examination we found a chronic otitis media with signs pointing to an intracranial complication. On the operating table a cerebellar abscess was found.

Neither of these patients was sent to the ear department by their medical attendant. Proper and timely treatment would probably have saved them both.

The patient with an ear discharging for years, or even for only a few months, if following one of the infectious diseases such as scarlet, measles, or influenza, does not usually appreciate its potential danger. He usually has no complaint beyond the disagreeable and often foul discharge. Only when headaches or dizziness begin to trouble him or when fever sets in, in other words, when a complication occurs, does he seek medical advice. Frequently a labyrinth involvement is mistaken both by the patient and his physician for some stomach disorder on account of the vomiting, the dizziness being taken for weakness following the severe gastric disorder. Sometimes the physician, in the course of a general physical examination of a patient suffering with some ailment apparently not at all connected with the ear discovers a chronic otitis. The practitioner must pay more attention to this finding and, if he is unable to take care of it himself, he should refer his patient to one who is alive to all modern methods of otological diagnosis, in order to decide what relation the chronic otitis has to the patient's present trouble, or what prophylactic measures to take in order to prevent a possible serious complication. It goes without saying that the modern otologist must have at his command a proper knowledge of the patient's general condition in order to estimate correctly his otological findings with relation to any other pathological processes.

As to the chances of a complication in any given patient, it, of course, involves a thorough study and classification of each individual case. This is based primarily on a study of the otoscopic picture, supplemented by a thorough examination of the nose and throat, and followed by careful observation and decision as to amenability to conservative treatment.

Otoscopically we must determine:

1. The character and amount of the discharge.
2. The seat of the perforation.
3. The presence or absence of granulations, polyps and particularly cholesteatoma.
4. The presence of an acute exacerbation of a chronic suppuration.

To facilitate the location of the drum perforation, we arbitrarily divide the drum into four quadrants by the handle of the malleolus which forms the vertical line, while another imaginary horizontal line is drawn at right angles to it touching the lowest point of the handle, or umbo. This divides the drum into: antero-superior, postero-superior, antero-inferior, and postero-inferior quadrants. Besides the drum proper or membrana tensa, perforations not infrequently occur in the membrana flaccida or Schrapnel's membrane for which every case must be examined for, as Alexander points out, "No otoscopical picture is complete without the examination of this membrane".

A perforation in the postero-superior quadrant or in Schrapnel's membrane usually denotes disease of the attic or antrum or, more commonly, both. From there, bone destruction, particularly when cholesteatoma is present, extends to the tegmen tympani, and when the latter is destroyed, the dura and its contents are exposed to infection. Sometimes a little scab or crust over this part of the drum may show, when carefully removed, a very slight discharge coming from high up, not at all known by the patient and easily overlooked by the careless examiner. Frequently a chronic headache is due to a damming up of such a slight discharge and will be promptly relieved on removing this lit-

tle scab, which thus clears up an otherwise difficult diagnosis.

A perforation in the antero-inferior quadrant generally means a tubal suppuration; the discharge is obstinate but usually clears up after paying proper attention to affections of the nose and throat, such as diseased tonsils, adenoids, hypertrophied turbinates, deviated septum, or chronic sinusitis. Such a suppuration, however, is never dangerous as far as intracranial complications are concerned but if not stopped it will lead to further destruction of the drum and hearing.

A perforation in any other part of the drum, no matter of what size or shape, as long as it does not extend to the margin of the bony annulus tympanicus, means a mucous membrane inflammation of the middle ear cavity and has little significance as far as intracranial complications are concerned, but if allowed to continue it will play havoc with the sense of hearing.

Our otoscopic findings should always be verified by the gentle and careful use of a probe to remove any suspicious scabs, and also to determine whether a drum perforation leads to any neighboring cavity, the direction of such a fistula, and its contents. Frequently a probe or an attic syringe, carefully used, in a marginal perforation, will bring down small whitish masses of cholesteatoma. These are pearly-gray masses of cast-off epithelial scales arising from the epidermis of the external canal, which proliferate into the middle ear through the perforation and by pressure destroy the surrounding bone, thereby exposing the dura and sinus to infection. According to Bondy, the presence of such masses of cholesteatoma, which can be suspected by an odor suggesting that of foul cheese, is in itself an indication for the radical mastoid operation, particularly so when symptoms of some intracranial complication are present.

An acute exacerbation of a chronic otitis is a very important finding for, while in a chronic otitis the disease is localized and may, as said before, expose some vital structure, it is the acute exacerbation with the superimposed inflammation of all the cavities surrounding the middle ear that actually causes the complication. This acute exacerbation can be recognized by observing the purulent discharge in the external canal which pulsates in an acute exacerbation but never in an uncomplicated chronic otitis.

A word concerning the polypi that occur in some chronically discharging ears. Although their removal with the fine snare is comparatively simple, the text-books do not emphasize the care that is required in their handling. Cases of labyrinthitis and meningitis following the simple removal of an apparently innocent aural polyp are not at all uncommon. Such an accident is due to the fact that the polyp sometimes originates from the bony capsule of the labyrinth and on removing it, the membranous labyrinth is torn into. It is therefore of extreme importance that the origin of the polyp be ascertained, and that great care should be exercised in its removal. A polyp arising from the labyrinthine capsule will give a fistula symptom. A simple way to produce this symptom is to press with the finger on the tragus of the affected ear so that the external canal is entirely obstructed. If an oscillation of the eyes or nystagmus occurs, sometimes accompanied by nausea and vomiting, leave the polyp alone. In the removal of aural polypi be sure that the snare cuts through the base of the polyp, the snare coming out clean and the polyp removed afterwards with forceps. This is unlike the procedure in the removal of nasal polypi where incomplete severence of the polyp with the snare is preferable so that a little traction can be employed thereby removing its base.

TREATMENT OF CHRONIC OTITIS

The ear must be gently but thoroughly cleansed in order to locate the perforation, thus to classify the case, and also as an absolute and necessary step in each treatment. The use of the Siegel otoscope is serviceable to aspirate pus from the middle ear, and to locate minute perforations. The nose and throat should be carefully examined, after cocainization, if possible, for any pathological lesions. This is particularly important if the perforation is central or in the lower part of the drum. If associated with chronic inflammation of the Eustachian tube, washing the tube with normal saline or bicarbonate of soda solution, through the Eustachian catheter, will frequently clear up a very obstinate otorrhea.

The more serious cases, however, are those with marginal perforations, cholesteatoma, or perforations in the upper part of the drum. These cases often tax the patience of the physician as well as that of the patient. When cholesteatoma is present it must be removed though not necessarily in one sitting. I have frequently removed pieces as large as the terminal phalanx of one's thum, although it was not even seen on the first examination. The use of an attic syringe is of great service in such cases. Recently I removed two balls of cotton from the middle ear inserted by the patient more than six months previously and entirely forgotten; the discharge cleared up very soon afterwards. Polyps and granulations are carefully removed. An alcohol swab, after thorough cleansing, followed by a dry swab insures, for the moment at least, a dry ear. A dry gauze packing is then inserted which must extend deep enough to touch the drum and the perforation. The packing must be changed every other day, always after careful cleaning of the canal. No drops of any kind are prescribed, and

the patient is instructed not to remove the packing and to avoid letting water enter the ear. Recently Lederman advocated the use of the Sulzberger iodine powder in the treatment of chronic otitis. It unquestionably has a great deal of merit, particularly so as it does not cake, as other antiseptic powders do, and thereby prevent the escape of pus. I have found it satisfactory in that it stopped the discharge leaving a dry perforation. The dry packing treatment, however, seems to have the advantage that it dries up the discharge by capillary drainage, and causes the epithelium of the external canal and edges of the perforation to bridge over and close the drum perforation. This causes the hearing to become almost normal, and prevents reinfection of the middle ear by the entrance of water through the dry perforation. The main point, however, in either of these methods of treatment, and I have frequently found it advantageous to combine them, is the thorough cleansing of the ear at each sitting, the sidetracking of any pathological findings in the nose and throat, and the frequent and close observation of the patient while under treatment so that the physician is on the spot, ready to prevent intracranial complication or operatively to interfere as soon as signs of it arise.

Miss S. S., 22 years old, came under my observation in June, 1923, with the history that the right ear was discharging since early childhood. For the preceding year she had been having frequent attacks of dizziness, particularly on bending down. Examination showed a chronic otitis media, perforation in the upper posterior quadrant, which on probing disclosed large masses of cholesteatoma. The discharge had a very foul odor. Hearing was markedly diminished, and a fistula symptom was present, indicating destruction of part of the bony labyrinth. Cleaning the ear followed by dry packing every other day, stopped the discharge, closed the perforation, caused the disappearance of the dizziness and brought the hearing to almost normal within six weeks.

G. P., age 46, had a double chronic otitis media since 16 years of age. He came under observation because of a furuncle of the external canal. He was almost deaf. Within two months both ears were dry; the perforations were closed; and he could hear ordinary conversation. In this case I first used alcohol drops instilled by the patient on top of the packing, and when the furuncle subsided, the packing was continued, but the drops were stopped.

G. J., age 36. Double chronic otitis for 20 years with marked diminution of hearing and kidney shaped perforation in each ear. Dry packing dried the ears and closed the perforation in two months.

EDITORIAL NOTE

The group of articles on subjects of practical interest that are presented in this issue (and to accommodate which the usual editorial departments have been omitted) comes from members of the staff of one institution—The Hospital for Joint Diseases, New York, which opened its fine new buildings a few months ago. These occupy a block front, facing a park, and present several novel features in planning and equipment. Especially interesting is the provision for every bed, whether in ward or private room, of a corresponding outdoor space on a shaded verandah, into which it can be rolled.

Founded about twenty years ago by Dr. Henry W. Frauenthal, the institution is also unique, we believe, as a large hospital, planned and conducted as a philanthropy, and created through the energy of one physician and his ability to attract support.

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American Journal of Surgery

Published by

SURGERY PUBLISHING CO.

Established by J. MacDonald, Jr., M.D.

WALTER M. BRICKNER, B.S., M. D., F.A.C.S., New York City, Editor

PUBLICATION OFFICE: 425 CARROLL STREET, ELMIRA, N. Y.

ADMINISTRATION OFFICE 15 EAST 26TH ST., NEW YORK, U. S. A.

where all communications intended for the Editor, original articles, books for review, exchanges and business letters should be addressed.

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